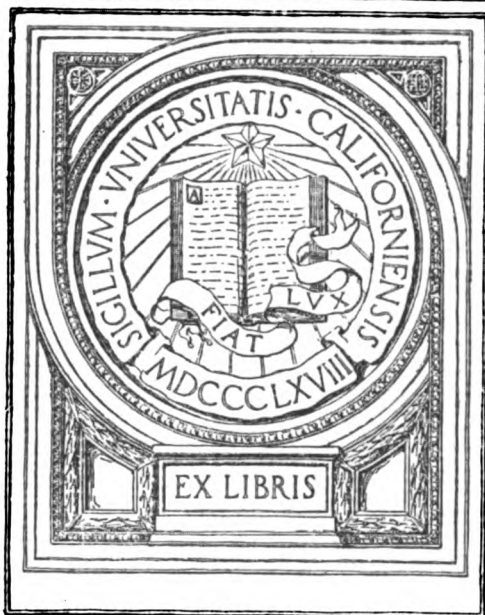




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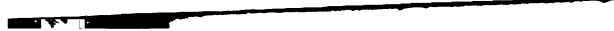


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# GUY'S HOSPITAL REPORTS

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# THE BICENTENARY OF GUY'S HOSPITAL

A SERMON PREACHED BY

HUBERT M. BURGE, D.D., BISHOP OF OXFORD

AT THE COMMEMORATION SERVICE

ON JANUARY 6, 1925, IN ST. SAVIOUR'S CATHEDRAL,  
SOUTHWARK

St. Matt. xxv. 40: "And the King shall say, Verily I say unto you, Inasmuch as ye have done it unto the least of these my brethren, ye have done it unto me; inasmuch as ye have not done it, ye did it not to me."

So St. Matthew's Gospel closes the record of our Lord's teaching: that teaching is rounded off with the Vision of Judgment, but judgment, you will notice, not upon mankind, but upon those who call Christ their Lord and King. The religion of Christ claims of all who profess it that they shall prove their love of God by the love and service of fellow-men, and further declares that as we are brought within the range of God's Great Love of us through Jesus Christ our Lord, we shall be inspired to reveal to mankind His Love through our own love and service—"If a man love not his brother whom he hath seen, how can he love God whom he hath not seen?" but, "Herein is Love, not that we first loved God, but that He first loved us: Brethren, if God so loved us, we must needs love one another." Our fellowship with men is given its true meaning and motive through our fellowship with God in Christ.

That is the distinctive mark of Christianity. If Christianity, Christian Service, is distinct from philanthropy, it is distinct just because the Christian is bidden to recognise that the service of fellow-men is a duty he owes primarily not to them but to Christ Himself. We are to go beyond the motives of compassion and human sympathy and kindliness of heart. We recognise in fellow-men not merely brothers but brothers for whom Christ died. So we Christians are called to lift this our bounden duty and service to that highest level where Christ can claim it, and consecrate our offering and use it for the highest ends of human welfare.

It is indeed a high calling: because it is so high, therefore, the failure to fulfil is the more humiliating. Listen to the unsparing condemnation: "Inasmuch as ye have not done it

to these, the least, to Me ye did it not : to Me ye who confess My Name have proved disloyal." For us, we are plainly told, there is never any room for the plea that the needs and interests of the least of mankind, the sick and the poor, the afflicted, the destitute, the prisoners, the fallen, are no concern of ours : that we have not the time, the means, the opportunity, the special disposition to be engaged in, or even interested in social well-being ; that it ought to be the concern of the State to make provision for the needs and the expansion of our Hospitals by a compulsory tax. Christ claims the free and willing service of these the least of His brethren from all who bear His Name. We offer it with all the loyalty of our hearts.

It is indeed fitting then that we should gather here in this Cathedral Church, linked by ties, new and old, with the Hospital of Thomas Guy, to offer our tribute of praise and thanksgiving for the service it has rendered these two hundred years to the least, it may be, of Christ's brethren in Christ's Name : not least of all, our thanksgiving for the spirit of simple Christian service which called out the bounty of Thomas Guy, and enabled this work to be begun and to grow and to win its signal place in our national life and tradition.

Single-hearted Christian Service indeed. Here is an instance, conspicuous enough, but one among many instances in our history, we English people may proudly say, of plain Christian citizenship. Thomas Guy, the son of Thomas Guy, a carpenter, who owned a wharf close by London Bridge. Thomas Guy, the son, apprenticed in the booksellers' trade, by his industry and intelligence takes the opportunities which good fortune brings him, prospers, and, as he prospers, thinks not less but more of opportunities of serving fellow-men, which prosperity offers him. Witness the almshouses he himself built and endowed at the age of thirty-five in the town of Tamworth.

Good fortune brings him good fortune : in the course of his long life, by skill and prudence and enterprise he amassed considerable wealth : again, not for himself, but to share with fellow-men. First he must devote it to the needs of the old hospital that stood at the corner of St. Thomas' Street across the road ; then came the great project, dearest of all to his heart, to acquire the site and build and endow the hospital where Guy's now stands.

So at the age of eighty years the old man passed away on December 27, 1724 ; and ten days later, to-day two hundred years ago, Guy's Hospital was opened to admit sixty patients.

The foundations of that work were well and truly laid. These two hundred years tell the story of steady ordered growth. In

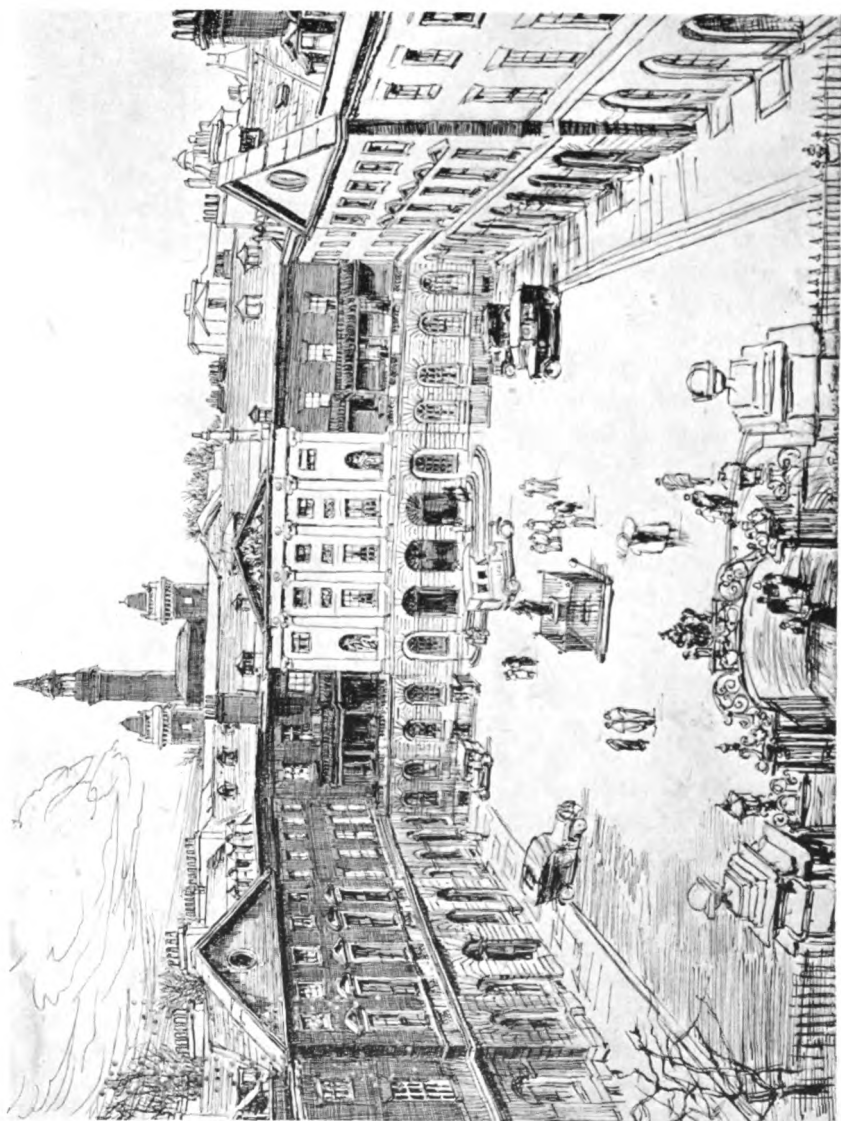
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GUY'S HOSPITAL, JANUARY 6, 1925.

By HANS LIP FLETCHER.

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each generation new needs have arisen, and new men have come forward, and have been moved to meet them, thankful to take their place side by side with Thomas Guy and to further his high purpose.

So, too, to-day the name of the humble bookseller's apprentice is linked with that noble line of our great men of medical science—Jurin and Saunders and Babington and Bright and Moxon and Hilton Fagge and Addison and Gull and Wilks and Golding Bird and Sharp and Astley Cooper and Aston Key and Hilton—who in their turn loved to honour the Founder and have always been the first to recognise the debt which medical science owes to the bounty of Thomas Guy.

We cannot doubt that the historian in future days will dwell upon two of the distinguishing marks of our own lifetime. First, the wondrous development of scientific knowledge applied to the treatment and cure of disease, the alleviation of human suffering, the saving of human life, the skilful nursing of the sick, not least of all, the provision for the advance of medical science and surgical skill by the careful training of young students in our great medical schools.

All honour to the men and women of our day, physicians, surgeons, nurses, for the use they have made of their God-given talents.

Shall our thankfulness for what they have given us be satisfied with the homage of our words? Shall their service be crippled, nay stultified, because it can be said that the large-heartedness, the thankful, generous spirit of Thomas Guy is no longer to be found among us, is stifled by the fever and the fret of modern England?

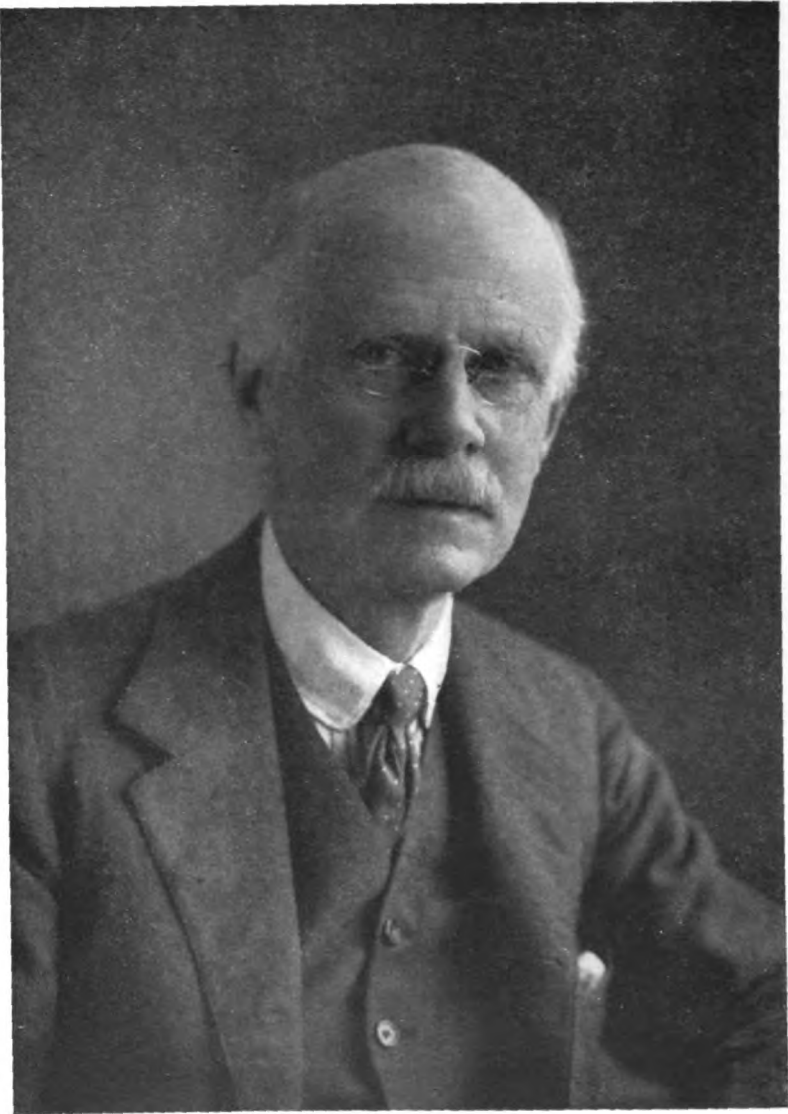
Then I think of that other feature of our age upon which the historian will lay stress; the accumulation of great wealth, due, no doubt, to great abilities, financial skill, enterprise, and sleepless industry, till fortunes which in my boyhood were unique, are now the commonplaces of prosperous and successful careers.

And we shall take our place, gladly and proudly, in the line of our best tradition when it shall be said of our own generation that loyal-hearted, thankful men, true to type, saw to it that their wealth was saved for the service of their fellow-men, for the sore needs of these the least of Christ's brethren.

It is easy enough to take Guy's Hospital as a matter of course: to discern that it stands for something in our common life which we could ill afford to be without. Stands for something! Stands for what? Stands there amidst the throngs and thoroughfares of crowded, pitiless London, where the individual

is so easily led to believe that he can count for nothing amidst those teeming multitudes, that everything matters but nothing matters much. And there Guy's Hospital stands, as the generations of men come and go, to bear its witness to those simplest, deepest truths our Heavenly Father would have all His children learn and teach, that human life is a sacred thing, that the highest duty is always possible, that self-sacrifice is sweet, and the love and service of fellow-men is the crown of life.

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Lauriston Elshaw

## IN MEMORIAM

LAURISTON E. SHAW, M.D., F.R.C.P.,

CONSULTING PHYSICIAN TO GUY'S HOSPITAL.

By JOHN FAWCETT, M.D., Senior Physician, Guy's Hospital.

LAURISTON ELGIE SHAW was the fourth son of Dr. Archibald Shaw of St. Leonards, and was born in London in 1859. Dr. Archibald Shaw had moved from London to St. Leonards on account of his health, and, although he suffered all his life from severe attacks of hæmoptysis, he lived to the age of seventy-six years, succumbing to an attack of cerebral hæmorrhage. His son Laurie was not equally fortunate.

Shaw received his early education at the City of London School, then situated in Milk Street, Cheapside. Dr. Abbott was the headmaster, and there for some time Shaw had as form-master Mr. Rushbrooke, the late headmaster of St. Olave's Grammar School. He remained at the school from January 1870 to Easter 1876, and shortly afterwards went to University College, from which institution he passed the Preliminary Scientific M.B. (Lond.) examination. He entered the School of Guy's in 1877, qualifying as M.R.C.S. in 1881. In 1882 he gained the Treasurer's Gold Medal for Clinical Medicine. In 1883 he obtained the M.D. degree of London University, and became an M.R.C.P. (Lond.) in 1885. In 1892 Shaw was elected to the Fellowship of the Royal College of Physicians of London.

Shortly after obtaining his degree, Shaw had his first attack of hæmoptysis, and on finishing the appointment of house physician, he went a voyage as ship's doctor on the *Sobraon*, one of two sailing ships which took patients to Australia in order that they might avoid the English winter, a favourite form of treatment in those days for pulmonary affections. Shaw had in mind the possibility of settling in Australia on account of his health, but he did not like the country, and on receiving a cablegram from a friend that an unexpected vacancy had occurred at Guy's, he decided to return to England.

In 1885 Shaw was appointed Demonstrator of Biology under Dr. Brailey, who was at the time the lecturer on the subject. In December 1886 he became Medical Registrar, and occupied

this post until his appointment in 1889 as Assistant Physician to the Hospital, and Curator of the Museum. From 1893 to 1901 Shaw was Dean of the Medical School. In due course, in 1907, he was promoted to the full staff, and retired at the age of sixty in March 1919, when he was appointed Consulting Physician to the Hospital.

In March 1892 Shaw married Miss May Spalding; their two sons were born in St. Thomas's Street, Reginald in 1893 and Maurice in 1894. Reginald became an officer in the 2nd Royal Sussex Regiment early in the war, and to the great grief of all who knew him was killed at Richebourg l'Avoué on May 9, 1915. Maurice served throughout the war, and having recently held the Radcliffe Travelling Fellowship of the University of Oxford, he is now following in his father's footsteps at Guy's in the position of Medical Registrar.

Mrs. Shaw was a most active and wise helper to Dr. Shaw throughout their married life. As an example of the many ways in which they did sterling work for Guy's, one of the most prominent was the foundation of the Ladies' Association, a society which continues to flourish and expand, and has been copied almost in its entirety by other hospitals in London and elsewhere. Mrs. Shaw acted as Honorary Secretary from the commencement in 1895 until 1919. The Salomons Infant Welfare Centre was another object in which both of them were deeply interested; they both were members of the Committee.

When Curator of the Museum, Shaw, with Sir Cooper Perry, undertook the arduous duty of revising the catalogue. For several years he devoted the greater part of his spare time to this work, which is incorporated in the first three volumes of the third edition. The task was very congenial to him, not only because it involved work of great value to the students and to the Hospital and School without bringing the author of it prominently before the public eye, but for the reason that the subject was of much interest to him personally and because he was convinced that in a large undergraduate school the specimens in its museum should be described in the main from the clinical aspect. Hence each specimen is accompanied, whenever possible, by a short clinical account in order that the student might acquire a mental picture of the patient and so a more vivid impression than would be the case from a description of so much dead matter. It is to be hoped that this living interest of the physician and surgeon may be secured in all future revisions of our catalogue, thereby setting the seal upon the invaluable work of the earlier editors, Hodgkin, Wilks and Habershon, as well as that of Shaw and Sir Cooper Perry.

In Volume XLVIII. of the *Guy's Hospital Reports* Shaw wrote a valuable paper in conjunction with Sir Cooper Perry on "An Examination of Fifty Cases of Malignant Disease of the Stomach," the specimens being all taken from the shelves in our Museum. Among other details they point out that colloid degeneration appears to be much more common in spheroidal than in cylindrical carcinomata and that the diffuse infiltrating type of growth is usually spheroidal-celled. In 70 per cent. of the cases the pylorus was involved, and in only two cases did the lesser curvature escape. A point of considerable interest in this paper is in connection with growths limited to the cardia. They quote Hilton Fagge as writing in the first edition of his *Principles and Practice of Medicine* that "almost all the cases which have been set down as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus extending into the adjacent part of the stomach." The authors came to the conclusion that Fagge was mistaken, and that in reality the growth begins at the cardiac end of the stomach and secondarily spreads into the gullet, for the reason that the three specimens of growth in this situation were all spheroidal carcinomata, whilst more than thirty specimens of malignant growth from all parts of the œsophagus, with the exception of two cases of sarcoma, were all proved to be squamous epitheliomata, a conclusion which nowadays is generally accepted as correct.

In Volume L. is another interesting paper by the same authors on "Diseases of the Duodenum," based upon the systematic records of post-mortem examinations at Guy's Hospital from 1826 to 1892, the specimens in the Museum, and the literature of the subject. The paper gives a very complete summary of the pathological conditions found in the duodenum, and it is particularly interesting to read of the cases in which burns and other sources of "septic" absorption are associated with ulceration, in view of the importance nowadays laid upon "septic" infection as a factor in the production of duodenal ulceration, but not so widely accepted when this paper was written. This paper ends with a very complete appendix, giving details of the 334 cases on which it is based. As Sir Berkeley Moynihan says in his book on *Duodenal Ulcer*, "there is no better presentation of the subject from the pathological standpoint, and the whole work is a monument of industry."

In connection with Shaw's work with Sir Cooper Perry it is pleasing to recall the remarks he made at the presentation to Sir Cooper on his retirement from the post of Superintendent of the Hospital in January 1920. Dr. Shaw said that he was



told he had been chosen to make the presentation because it was believed that he was Sir Cooper's oldest friend at Guy's and had been more closely connected with him in his work than anyone else. He then referred to the commencement of their friendship thirty-three years before, when they were applicants for the vacant post of Assistant Physician, and Sir Cooper, then of the London Hospital, was selected. Dr. Shaw ended by saying, with his usual unselfishness and truth, that he had never for a moment ceased to be profoundly grateful to the Governors for postponing his election and securing for him one of the longest, closest, and happiest friendships of his life.

In Volume LVI. of the *Reports* Dr. Shaw gave details of one of the earliest recorded cases belonging to the group of so-called "Splenic Anæmia," in which the chief feature was severe recurrent hæmatemesis, to which Sir William Osler had already drawn attention. The spleen was much enlarged when hæmatemesis first occurred, but for how long it had been so is unknown, nor was there any obvious anæmia preceding the attack of hæmorrhage. Shaw discusses the question of surgical treatment of the condition, which had at that time only been attempted on a few occasions, and regrets that owing to the uncertainty of diagnosis he did not advise splenectomy in this case.

Shaw also wrote a few other articles on medical subjects in Quain's *Dictionary of Medicine* and in *Brain*, but his later writings were mainly confined to addresses on the medical and social problems in which he was so deeply interested; the following is a list of some of the most instructive:—

"A Contribution to the Study of the Treatment of Hospital Abuse": *Lancet*, Dec. 3, 1904.

"The True Aim of a United Medical Profession and the Handicap of the Trade Union Bogey": *British Medical Journal*, July 16, 1910.

"The Place of the Hospital in a Civilised Community": *Guy's Hospital Gazette*, 1911.

"Medicine and the State": *Lancet*, July 20, 1918.

Of Shaw's invaluable work as Curator of the Museum I have already written; equally valuable in another direction was his work as Dean. Throughout the whole of his tenure of office at Guy's, the prosperity of the School and of its students was uppermost in his mind. He was never tired of talking of them and of their interests. Nothing was too difficult for him to attempt if he thought it was for the advantage of the School,

and during the eight years in which he acted as Dean he devoted himself, heart and soul, to its welfare. Living in St. Thomas's Street until 1900, he was enabled to keep in close touch with everything which was going on, and so to make full use of his powers of organisation and to exert a very efficient and direct control over the work in the Dean's office, with consequent great advantage to the School.

As Dean, among the many objects to which he devoted his skill and attention, the erection of the new school buildings



From *The Guyoscope*, June 9, 1897.

was one of the foremost. With the consent of the Governors, the physiological building, the first part of the recently completed new Medical School buildings, was erected at a cost to the School of £13,000. The money was raised by a loan or collection from the Staff, the repayment of the loan becoming a charge upon the students' fees, so lessening the small remuneration already received by the majority of lecturers and demonstrators. The building was opened by H.R.H. the Prince of Wales, later King Edward VII, in May 1897.

In the following year Mr. A. J. Balfour, who had been recently appointed a Governor of the Hospital, distributed the prizes and delivered a most interesting address in which he initiated

the appeal for the endowment of medical education and research, from which the School has benefited in many ways,—by the beautiful Wills Library, the Gordon Museum, the Sir William Dunn Lectureship on Pathology, and also by many most generous gifts of money from the Governors and others, in particular by the bequests of one of our late surgeons, Mr. L. A. Dunn, and that devoted servant of the Hospital and School, the late Mr. C. H. Wells.

No record of Shaw's activities at Guy's, manifold as they were, would be complete without reference to the subject of the "concentration of common courses of instruction for the preliminary and intermediate subjects of the medical curriculum at one or more centres in London." Shaw believed wholeheartedly in the economical and educational value of "concentration," and as Secretary of the Concentration Committee of the University of London he did an immense amount of work in conjunction with Sir Henry Butlin, then Dean of the Faculty of Medicine. In 1901 the School had, chiefly at Shaw's instigation, expressed its approval of the general principle, but in 1903 that approval was withdrawn. It was he, too, who revived the Conference of Deans in order to promote closer co-operation between the Metropolitan Schools. Other changes which took place during his term of office as Dean were the re-constitution of the Dental Department and the separation of its funds into the two classes as they still exist,—the Dental School Fund and the Dental Hospital Fund. The foundation of the registered list of householders with suitable accommodation for students was also laid by him.

Shaw was an excellent clinical teacher, most attractive perhaps in his earlier years as Medical Registrar, and as Assistant Physician in the Out-patients' Department where many generations of Guy's men have cause to remember and to be ever grateful for the sound methods of examination which he employed, the lucidity of his inquiries and comments, with their telling quiet humour, common sense and practical humanity.

Although ever ready to discuss any theoretical explanation of a case, he always tried to pin down himself and the students to make up their mind whether there was a lesion of any kind, what was its position and how it was to be treated. He hated the use of loose general terms like "toxæmia," regarding them rightly as leading to "quackery" in medicine, to self-satisfaction in slipshod methods of mind and of examination, and consequently to defective treatment.

In Shaw's clinical lectures the same features manifested

themselves. The salient details of the case were selected and presented in so clear and orderly a manner as to surmount the difficulty and make it seem as simple as a complicated abdominal operation by a great surgeon. Take, for example, the introduction to a clinical lecture on "A Case of Persistent and Grave Diarrhœa," published in the *Guy's Gazette* for May 22, 1897, or, again, a clinical lecture in the following year on "Ascites," which contains a good example of his critical humour: "There is a method of diagnosis adopted by some out-patient clerks and occasionally with success. They start by saying, 'Do you drink much tea?' and so till they gently lead the conversation up to gin; if the patient acknowledges to drinking gin they at once conclude that he has got cirrhosis and so ascites." Again, no student nor medical man could even now desire to read a more helpful lecture than that on "Some Points in the Treatment of Phthisis," in the *Guy's Gazette* of September 1, 1900, an example of clear thinking and exposition based for its application on the possession of the requisite knowledge and skill to recognise the disease in its early stage.

These illustrations from his clinical lectures were, like his bedside teaching, full of common sense, of practical wisdom and clinical acumen. To deal with the case from the standpoint of diagnosis was by no means sufficient for him; he was constantly impressing upon the students the importance of prevention, pointing out to them how wasteful it was to get a man into hospital and afford him temporary relief, and then discharge him with nothing but a bottle of medicine and without any instructions for conducting his life in the future in order to help him to guard against a relapse into his former state. The guiding principle of Shaw's medical life, that the individual medical man was the first line of defence in the prevention of and attack upon disease, was reflected invariably in his teaching, namely, that the students must not only learn to recognise and correctly interpret the signs of disease, but also as of equal importance to educate themselves in the right method of eliciting the details of the illness and of its earlier manifestations, and any features of importance in the life history of the patient.

The demonstrations which he gave in Pathology in the Museum for several years were most attractive and instructive, and drew large classes to hear them. He was the first member of the Staff to give revision classes in "medicine," and this he continued to do for several years with most successful results.

The last great work performed by Shaw for the Medical School

was as Chairman of the Reconstruction Committee in 1918, when an inquiry was instituted into the staffing and teaching of all departments, and a most voluminous and valuable report, in large part founded on his suggestions, was drawn up, and approved by the School in 1919.

Shaw was an excellent Chairman of Committee, of which his colleagues had evidence during the time in which he occupied the position of Senior Physician. The business was conducted in an orderly and clear manner, and everyone who wished to express his opinion, whether junior or senior, was given full opportunity to do so within fairly wide limits. As Chairman of the Staff meeting and of the Medical Committee in particular his genial and businesslike attitude was highly appreciated by his colleagues.

This summary of Shaw's lifelong work for Guy's is by no means complete, and there are still two more subjects which must not be omitted,—the formation of the Clubs Union by the amalgamation in 1891 of the various students' clubs and societies which then existed as separate units, and the purchase of the Club Ground of nine acres at Honor Oak Park. Both of these schemes were due largely to Shaw's initiative and driving power, to his appreciation of the needs of the students and of the desirability of improving the status of the constituent clubs. He was the first Honorary Secretary of the Clubs Union and held the post until 1894. He was President of the Clubs Union in 1900 and again in 1921 and 1922.

Outside Guy's his activities were many and varied. The social and ethical problems of professional life appealed strongly to him, and in this connection he worked hard for many years for the British Medical Association until, in 1912, the dispute over the National Health Insurance Act brought it to a tragic close.

Dr. Shaw had long been a member of the British Medical Association. He was a vice-president of the Section of Medicine at the Annual Meeting at Ipswich in 1900, and was one of the general honorary secretaries of the Annual Meeting in London in 1910. He had also held the office of president of the Metropolitan Counties Branch and was long its treasurer. He became a member of the Central Council in 1907, and continued in office until 1912.

Sir Jenner Verrall wrote the following critical and appreciative account of Shaw's work for the "Association" in the biographical notice in the *British Medical Journal*, January 5, 1924.

"An obituary notice of Lauriston Shaw, certainly one in

the columns of the *Journal*, must give some account of his work in and for the Association. In time of peace and of storm it showed the man he was. The year 1900 and succeeding years up to 1911 covered the active period. Interested always in the social problems of professional life, his position in the medical school at Guy's Hospital and his private work brought him full knowledge of the medical man and his difficulties from student days onward. With this experience as a guide he filled many important posts in the Association: as an officer at various annual meetings, as president and treasurer of the Metropolitan Counties Branch, and, finally, as member of Council from 1907-8 to 1911-12. It was, perhaps, as chairman of the Central Ethical Committee that his gentle method and charm of manner were most noticeable. Shaw had, from temperament and experience, formed the opinion that the State should concern itself with the medical care of the employed population in general, and that this must involve some system of contract with the medical profession. It followed that the National Health Insurance proposals found in him an advocate already convinced. The principle, however, was far from receiving acceptance by the profession, which would gladly have kept itself free of all the ties of contract service; nor were the conditions offered such as were considered by it fair and workable. It was bitterly, and not unnaturally, resented that Dr. Shaw, a consultant, should favour a system, held to be faulty, the burden of which would fall on the general practitioner.

"He knew full well that his support of so unpopular a measure must greatly injure, if not destroy, his position as a consulting physician. It was not in him to waver. In justice to him it must be remembered that, when once the State had moved in the matter of health insurance, some scheme would inevitably be adopted. So much for the principle. As to the conditions, including remuneration, it might fairly be argued that those first proposed were imperfect and inadequate. Further, it is just now especially clear that revision of these terms from time to time will be undertaken.

"There was a grim irony in the fate which brought him, chairman of the Central Ethical Committee, into strong collision with so large a majority of the profession. But surely those who felt most that he was wrong must also have realised that here was one who saw his ideals clearly and counted the world well lost for himself if so the ideal might be served. In that frail body, behind that quiet voice and rather pathetic smile, there dwelt a brave and unselfish spirit."

Dr. Alfred Cox, Medical Secretary of the British Medical Association, wrote as follows, in the same number of the *Journal*:

"He was a strong believer in the principle of insurance, and this led him not only to dissent from the action of the Special Representative Meeting in December 1912, which refused the offer of the Government, but to take part in steps for organising those medical men who disagreed with that decision. His action on that occasion led to his withdrawal from active work in connection with the British Medical Association, and many of his old friends profoundly disagreed with the line he took. But no one who knew him had any doubt as to the perfect honesty of the man, and those who were intimately associated with him realised what a great sacrifice he had made. I do not think that I have ever known any man who was more devoted to the Association than he was, or to whom the breaking off of active work in connection with it could have been a greater sacrifice. He had filled with great distinction one of the most difficult positions in the Association—namely, the chairmanship of the Central Ethical Committee—and was a member of many other committees; in fact he was one of the men who was always called upon when any new or particularly difficult problem arose, and he never declined the call. Although he dropped his active central work for the Association at the end of 1912, he never ceased to take an interest in what was going on, and he continued in many ways to do good work for the profession. He was one of the most persuasive speakers and writers I have ever known. I particularly remember his masterly defence of the private practitioner against the idea of a whole-time State Medical Service. His broad and philosophic knowledge of general politics made him particularly useful in medical politics. It was a great privilege to be associated with such a man, and I shall always remember him gratefully for his affection for the Association, his sweet reasonableness in debate, his wisdom in counsel, and his invincible moral courage."

It may be here recalled that at the special Representative Meeting of the British Medical Association in December 1912, the members decided to reject the proposals of the Government and to decline to serve under the Act. Dr. Shaw believed that this decision was wrong and that it was his duty to combat it with all the force he could muster, and he therefore resigned all the offices he held in the Association. "As to the correctness of his views," Sir Ewen Maclean wrote in a letter to Mrs. Shaw in November 1924, "proof was soon forthcoming, inasmuch as at a further Special Representative Meeting in January 1918, a resolution was passed releasing practitioners from the pledge

previously given to the Association not to accept service under the National Insurance Act."

As far as the writer knows, Dr. Shaw never complained about nor indeed referred to his experiences in the controversy over the Insurance Act until June 1922, when a presentation was made to him and Mrs. Shaw by the members of the London Panel Committee, of which he was a prominent member. In returning thanks Dr. Shaw said, "I can never forget the time when I was, perhaps, one of the most unpopular persons in the profession, when, owing to my disagreement with some of my colleagues, I found nearly all the channels of my activity closed to me—political, educational and professional"—and he then proceeded to enunciate clearly his convictions on the subject of medical politics. "I should like to say I certainly disagree with the attitude taken that medical politics are no sort of politics, or derogatory, and that there is something less valuable in medico-political work than in other forms of medical work. If I should place the usefulness of our work as medical men I would put first, of course, discovery, research. Next I place our medico-political work that enables those researches to be made available for the whole community." Towards this end the "great experiment" of National Insurance was in his eyes of supreme importance, a plant of many years' growth, based on the grounds that he believed it to be impossible for the vast majority of the population to pay a doctor adequately unless they had previously made provision for illness, which the poorer classes systematically neglected to do through no fault of their own, and which they never could do adequately without some sort of State aid. He also believed that the labourer was worthy of his hire, and the greater the certainty of being paid a fair remuneration for work done the more attractive would the medical profession be to the right type of man, and hence the better preservation of the public health, a most important part of the duty of the State. In this contract service between Medicine and the State he believed that a real and lasting success could only be obtained with good general practitioners in the first lines of attack and defence. "These two great principles," he said, "the guiding principles of my medico-political life, I found enthroned in this Insurance Act."

I have referred at considerable length to this part of Shaw's career because it was one in which he spent so much time and energy. Like all politicians, especially in the case of leaders, he came in for much abuse often where least deserved. As Mr. Balfour said in his appeal in 1898 at Guy's for the endowment of medical education and research, "the members



of the medical profession have the good fortune of often seeing the results of their labour for the good of mankind rewarded before their eyes. To the politician is seldom given the opportunity to judge of the value of his labours or to estimate whether his work has been on the whole for the advantage of mankind." Shaw at any rate saw some of the results of his labours working out on the lines of his own convictions, and he had the whole-hearted support of a large number of his medical brethren. For those who abused him one could but regret that they did not know the man, for whether one agreed with him or not in some of his views, no one who knew him, least of all his many friends and colleagues at Guy's and elsewhere, could doubt for one instant his sincerity and honesty of purpose and complete absence of self seeking; nor could they fail to recognise the value of his ideals in the practice of his profession and the high standard he set for himself and others.

His interest in the question of National Health Insurance was none the less active after the reverse, if one may so call it, of 1912, which severed his official connection with the British Medical Association. His work on the London Panel Committee was very agreeable to him, and he laboured steadily at the subject for a further eleven years until compelled in October 1923 to resign on account of increasing ill-health.

The presentation to him by past and present members of the London Panel Committee in 1922 has been already referred to; the gifts he received and the manner of the giving were a constant source of pleasure to him and to his friends during the remainder of his life.

Dr. H. J. Cardale, the Chairman of the London Panel Committee at the time of Shaw's death, relates in an account of him in the *British Medical Journal* of January 5, 1924, how "Shaw was a member of the original provisional Panel Committee for London, and from the first held the responsible position of treasurer to that committee and to the Statutory Committee which followed it, to which position he was unanimously re-elected year by year. As treasurer he was *ex officio* member of the three standing sub-committees of the London Panel Committee, and was also invariably appointed to any special sub-committee. Always most regular in his attendance, he put in an enormous amount of work which was of the highest quality.

"Owing to failing health Dr. Shaw had wished to resign from the London Panel Committee in 1922, but he withdrew his resignation at the unanimously expressed desire of the members of the committee that he should continue, as and

when he could, to assist the committee with his advice. In October 1928, however, Dr. Shaw felt that his health would no longer permit of his further participation in what, I have every reason to believe, was congenial work. At the same time he resigned his membership of the London Insurance Committee, on which body he had been a representative of the profession, appointed by the Commissioners of National Health Insurance and later by the Minister of Health. His services as a representative of the London Panel Committee at the annual Conference of Local Medical and Panel Committees were of the highest value, and the Conference loses one of its most attractive and valuable personalities. His loss produces a blank almost impossible to fill. Shaw and his work will be held in affectionate and grateful remembrance by his colleagues on the London Panel Committee and by all insurance practitioners."

In June 1915 he was nominated by the Minister of Health to serve as a Manager of the Metropolitan Asylums Board, and here he devoted himself particularly to the provision of sanatorium accommodation for tuberculous patients and acted as chairman of the Pinewood Sanatorium Committee. Fond as he was of this side of his voluntary work, he was compelled by increasing ill-health to resign in October 1923.

What a mass of achievement is depicted in this outline of Shaw's life; how impossible in an article like this to fill in all the details; how easy to leave great gaps in descriptions of his life and character! Shaw was fond of a quiet social life, and nothing delighted him more than to have his friends around him. Many of his ward-clerks and "clinicals" will remember with pleasure the dinners at Harley Street, the tennis parties at Wendover, and what a delightful host he made; others will recall some of the happiest days of their lives spent with him in the walks which he loved, and in earlier days the journeys by bicycle before the advent of motor-cars, when even the country roads near London gave pleasure to the man with the "push bike."

Although during the war Dr. Shaw took to wearing a short coat, soft felt hat, and the soft collars of that gloomy period, most old Guy's men will recall him best and most fondly in his "tall-hat" days, especially in the white one of summer, with his never-failing companion, wet or fine, the umbrella on his arm. With his quick, active step and head and body swaying somewhat from side to side as he walked down the Colonnade or along St. Thomas's Street, the ready smile, the rapid blinking of the eyes, particularly when asked some question, eyes which seemed to take in all and sundry in the process, there was

never the least suggestion of invalidism about him, although perhaps he had throughout his life a greater experience of living on the brink of ill-health than, happily, falls to the lot of most.

Throughout his career at Guy's, Shaw suffered at intervals from attacks of hæmoptysis, one of the most severe occurring shortly after the birth of his second son in 1894, and after that he and Mrs. Shaw went abroad during four successive years at Christmas time, twice to Mentone and twice to Switzerland, these holidays proving of immense value in restoring him to health. To maintain his forces in good fighting order he always took care to lead as healthy a life as possible. We often saw him enjoying himself at tennis at Honor Oak Park; he was fond of swimming and boating on the Thames in his earlier years, and of bicycling, and throughout his life he was never more happy than when walking with members of his family or his friends, which he continued to do until four or five months before his death.

In November 1915 Shaw had a very severe attack of pneumonia and for a time his life was despaired of; he was so exhausted by this illness that he was unable to return to work at the Hospital until May 1916. Then he seemed to get a fresh lease of life and was in active work until 1923, when in the early part of the year he began to show signs of a reawakening of the disease which cast a shadow on his life. In August he became more seriously ill and had to take to his bed in the latter part of that month. From this time onward he became gradually worse and died on Christmas Day 1923.

Although not a strong man physically, Shaw never thought of himself. He fought his illness with the same vigour, self-restraint and unconsciousness of self which characterised his actions in the varied spheres in which he played his part. He was a reformer to his finger-tips, always cogitating some scheme for the benefit of humanity, the good of his medical brethren in particular, or for the welfare of the Hospital and School which he loved, and for which he did so much.

As the writer of the obituary notice in *The Times* said truly, he was transparently honest, tactful, a pleasant and persuasive speaker with the gift of quiet humour. As a speaker he was better in expounding his views in Committee than on the public platform. If the cause was in his opinion good—be it popular or otherwise—he would fight for it to the last ounce. Like many reformers, one would never have appreciated from his appearance how pugnacious and persistent he could be if the spirit moved him, with a pugnacity free from temper,

with a persistence mitigated by humour and tolerance, but becoming at once scornful in tone to arguments which breathed a flavour of misstatement or of subterfuge, of selfishness or of nepotism.

He is sorely missed, and will be long remembered by a host of medical brethren and of old students, and by his colleagues on the Staff of Guy's, all of whom realised to the full the unselfishness of a life whereby they benefited so greatly and which rendered his own career so happy.

Shaw could never subscribe to any particular form of religious faith, and yet at heart he was a religious man. In his earlier years his aspiration was to help his fellow-men, in the later to add from his experience the power to inspire others; surely two of the greatest moral influences a man can exert in the spring-time and autumn of a life, and when he died on that great birthday of Christmas there was no one but could justly and truly say of him, as Bunyan did of "Mr. Valiant-for-Truth," "So he passed over, and all the trumpets sounded for him on the other side."

# MASSAGE AND REMEDIAL EXERCISES IN MEDICINE

## INTRODUCTION

MASSAGE and remedial exercises hold a recognised position in surgery. In the treatment of medical conditions their place is less well established, and less attention has been paid by physicians than by surgeons to the indications for their use. There is in consequence a lack of co-operation between the physician and the masseur: the physician knows little of the details of massage technique, and the masseur but little of the principles which have prompted the physician to prescribe physiotherapy.

Progress in this method of treatment has therefore been slow; the recognised text-books on physiotherapy contain little more advice than that found in those written thirty years ago, and their teaching on medicine is that of the last century.

The Editors think that there is scope for a series of articles dealing with the indications for physiotherapy, and giving a general outline of such methods of treatment as experience shows are of real value in medicine. The forthcoming numbers of the *Reports* will contain articles on the use of massage and exercises in the treatment of (1) Circulatory Disorders (Dr. G. H. Hunt), (2), Abdominal Disorders (Dr. A. F. Hurst), (3) Pulmonary Disorders (Dr. G. H. Hunt) and Asthma (Dr. A. F. Hurst), (4) Nervous Disorders (Dr. C. P. Symonds), and (5) Diseases of Joints (Mr. W. H. Trethowan).

G. H. H.

## I. MASSAGE AND REMEDIAL EXERCISES IN DISEASES OF THE CIRCULATION

By G. H. HUNT, M.D., Physician to Guy's Hospital.

DISEASES of the circulation have been treated by massage and graduated exercises for a great many years in this country, at continental spas, and particularly in Sweden. Writers in books on massage and remedial gymnastics describe various treatments in considerable detail, but say little of the fundamental principles that should guide their selection for any

particular patient, and no distinction is drawn between the main types of heart disease. In the following account an attempt is made to assess the relative value of the different methods of treatment, and to describe the indications for their use.

In the Swedish system there are three main types of treatment, which are defined in the text-books as follows :

1. *Massage*.—The manipulation of the soft tissues of the body.

2. *Passive movements*.—The movements of the joints by an operator, or an outside force, the patient being completely relaxed.

3. *Active movements*.—Movements performed by the patient himself with or without the *assistance* or *resistance* of an operator or other outside force. (Words “outside force” are added, as weights are sometimes used to assist or resist in both active and passive movements.) Active movements are by far the most important of the three methods and will be considered first.

Muscular work may do good or harm in heart disease and is therefore a means of treatment which must be used with considerable discretion. In every case two questions have to be considered.

(1) At what stage should active movements be started ?

(2) What amount of muscular work should be given, and how quickly should it be increased ?

The answers to these questions will depend upon the nature of the case, and it is convenient to consider them in the three following types of circulatory disorder.

(A) *Acute carditis* : the condition in which the heart is the seat of a recent infection, such as rheumatic fever.

(B) *Chronic cardiac insufficiency* : the condition in which the heart is structurally damaged by some degenerative process, or by some inflammation which took place months or years before. A typical example is afforded by the woman of forty, who is admitted to hospital with cardiac failure from auricular fibrillation and mitral stenosis, the results of an attack of rheumatic fever in her youth.

(C) *Effort syndrome* : the condition in which the patient is suffering from a functional circulatory disturbance without organic disease of the heart or blood vessels.

The harmful results of excessive muscular work are absolutely different in the three groups. In Group A it may cause a recrudescence of the infection : this, rather than a general dropsy from cardiac failure, is the danger in young people, for cardiac failure rarely, if ever, occurs in children, unless

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the heart is the seat of some acute inflammation. In Group B, on the other hand, overwork may cause œdema and other signs of lost compensation, whereas the danger of a return of the original inflammatory trouble is a remote one. It is still disputed whether hard muscular work ever causes any serious damage to patients in Group C, but it is quite certain that its effects are never so disastrous as they may be in the two former groups.

Failure to realise the fundamental differences in these three groups is responsible for much of the confusion of thought found in many of the text-books on massage; thus we read in one standard work that "hurried breathing, perspiring, dilatation of the nostrils, yawning and palpitation are indications that treatment should be suspended." To take one only of these supposed contraindications, namely, "hurried breathing," exercises which cause this in patients in Group C are often beneficial; in Group B they are dangerous long before the breathlessness is of such a degree that dilatation of the nostrils occurs; in Group A they are infallibly disastrous.

The indications for rest and exercise are so different in the three classes that each must be considered separately.

### *Group A. Acute Carditis*

At what stage should active work be begun? The answer to this question will vary with the severity of the attack. In some children rheumatic fever runs a benign course, and within a few days of the onset all signs of acute inflammation of the heart and joints have disappeared. In such cases active movements can be started after four to five weeks' rest in bed. In other cases, however, progress is much less satisfactory. The heart may remain enlarged with a persistent murmur, or tachycardia or slight fever may continue; after months of complete rest no obvious change takes place, and we are forced to the conclusion that the cardiac damage is permanent. It is possible that rest and country air might restore some of these children to health, but the number of convalescent homes, whose authorities are willing to take rheumatic children with heart lesions, is so limited that many have to be treated in hospitals in the larger towns. The problem as to when to start active work is a difficult one in such cases. My own practice is to wait for three to six months after the condition has become stationary, that is to say, until it seems that no further improvement is likely to result from rest alone, and then to start graduated exercises.

What amount of muscular work should be given, and how quickly should it be increased? This must be judged by the effect of exercise on the heart, and the most convenient measure of this is afforded by the pulse-rate. In the plan adopted at Guy's Hospital, three rules are followed :—

(1) The scheme of treatment at first lasts ten minutes, and gradually this time is increased to thirty minutes.

(2) It consists of general massage and exercises; very little active work is done for the first three or four days, to allow the patient to get accustomed to the treatment. During the succeeding week it is slowly increased, but is still not sufficient to raise the pulse-rate more than two or four beats: after ten days' treatment, one or two exercises are included which are appreciably harder than the rest; these are known as the test exercises, and should be sufficient to raise the pulse-rate four to six beats; \* they are gradually increased in severity, until at the end of the fourth week they raise the pulse-rate eight to ten beats. The exercises required to produce the desired effect on the pulse have to be gradually augmented; at first flexion and extension of the smaller joints, such as the wrist and ankle, is sufficient; subsequently, the larger joints are moved, and the work is further increased by making the patient do each exercise a greater number of times. The work can be made harder still by the patient doing the movements against the resistance of the masseur. In patients progressing favourably it is usually found that the test exercise has to be increased in amount every two or three days, and the degree of the patient's improvement is shown by the increase required.

(3) The exercises are reduced or stopped altogether if there is any indication of a return of the inflammation. The signs vary in different cases, but generally, before any obvious changes in the heart develop, there is some alteration in the temperature or pulse. The former is easy to recognise; it may be more difficult to appreciate the latter at its true value, but it may give valuable information in two ways. In the first place, if the pulse-rate, taken from day to day, is gradually rising, it is an indication that cardiac damage is progressive. In drawing any conclusions from this, it is essential that the pulse-rate should always be counted under the same conditions; these are best obtained by taking the pulse-rate shortly before the patient wakes, for in this way the influence of excitement and

\* Directly after the completion of the exercise, the pulse is counted for a quarter of a minute, the number of beats multiplied by four, and the rise of pulse-rate calculated accordingly; thus if there are 19 beats during the first quarter-minute after exercise, the rate is reckoned as 76 ( $19 \times 4$ ), and if the pulse before exercise was 70, the rise is reckoned as 6 beats ( $76 - 70$ ).



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restlessness is reduced to a minimum. Secondly, it is important to note carefully the effect of the test exercise; if it is found that this has to be increased in order to raise the pulse-rate four to six beats, the condition of the heart is improving, but if no increase is necessary, and still more if the same test exercise, repeated day by day, causes a progressively greater rise in pulse-rate, it is a warning that inflammation has not completely subsided. The value of this sign is not sufficiently recognised, and the following case affords an example of this.

The patient, a young woman, was admitted to hospital with rheumatic fever and a dilated heart. After a period of rest in bed, and prolonged convalescence, she was re-examined, and the heart found to be normal and her exercise tolerance good. She returned to light work, and her exercise tolerance was tested every week; one day it was found to have deteriorated considerably, but as there was no other evidence whatever of any recurrence of the inflammation, she continued to work. Two days later she developed a second typical attack: the lowered exercise tolerance was the first sign of the recrudescence of the infection.

The amount of exercise of which the patient is capable guides us in deciding when to allow him to get up. (The actual movements done by the patient are described in the Appendix.) When he is able to do the easiest exercises involving the trunk muscles, he is allowed to get up on a couch; at the stage when he first starts dumb-bell exercises he is allowed up in a chair; and when he can raise 1 lb. dumb-bells twenty times he is allowed to start walking.

These rules are, of course, not universally applicable, and indeed they have been modified considerably from year to year, but experience shows that they are a useful guide to the masseur. Probably they err on the side of caution, for if they are followed, it is not often found necessary to reduce the amount of exercise; in some cases it is possible to allow a rather larger rise of pulse-rate in the test exercise, but I have seen a certain number of patients, where this has been done, develop a tachycardia which only subsided after all active work had been suspended for several days or even weeks.

*Massage and passive movements.*—These are of doubtful value in this group: possibly they improve the nutrition of the muscles of the arms and legs, but there is no evidence that they have any direct effect upon the heart itself.

### *Group B. Chronic Cardiac Insufficiency*

It is convenient to consider the treatment of cardiac failure at four different stages.

*Stage 1.*—That of acute failure. At this stage there is a varying degree of breathlessness even at rest; cyanosis may be present, the subcutaneous tissues are œdematous, and there may be fluid in one or more of the serous cavities. At this stage, rest is so essential that the patient is obviously quite unsuited for any treatment by exercise, either active or passive. A little general massage may promote sleep, but it is so essential to ensure this that it is better to rely on more certain means, such as hypnotics.

*Stage 2.*—At this stage the respiration rate has fallen to only slightly above the normal, and the dropsy is diminishing. Massage (kneading and effleurage) for the legs undoubtedly hastens the disappearance of the œdema, and I am quite convinced of its value in cases where this is unusually persistent.

Other methods of treatment are directed towards aiding the return of the blood to the right side of the heart, and so diminishing the venous stasis. Of these the most valuable are breathing exercises, for there is definite physiological evidence that they do promote this by increasing the negative pressure in the thorax. It is reasonable, too, to believe that they may open up bronchi clogged with secretion, and so increase the aerating capacity of the lungs. A second method used is passive movements to the limbs. It is difficult to obtain definite evidence of the effect of these. On theoretical grounds, however, we should expect them to aid the venous return, for they compress the veins, and the action of the venous valves only allows of a flow of blood towards the heart. Thirdly, massage of the engorged liver has been advocated; it is an organ so easily distended that it might be hoped that blood could be squeezed out of it mechanically, but I have never seen a case where I was satisfied that any definite diminution of its size was produced. More observations, however, are needed on this point.

*Stage 3.*—By this time the respiration rate is normal, and the œdema has gone. It is now safe to start active movements. As in Group A, the first three or four treatments are merely preliminary; but subsequently the rate of increase in active work is more rapid than in Group A, and by the end of ten days the test exercises raise the pulse eight to twelve beats (Scheme 2). A larger rise is allowed than in Group A for two reasons. Firstly, the signs of deterioration, for example a slight return of œdema, or a persistent quickening of respiration rate, are easier to appreciate than those of a recurrence of inflammation. Secondly, if the exercise given should have been excessive, the harm done is easily remedied by a few days' rest; whereas once

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inflammation is started again, it may progress for weeks in spite of all treatment. It is, of course, desirable in both groups to progress as quickly as possible, and reduce the time spent in bed to the minimum; experience shows that as a rule it is possible to increase active work more rapidly in Group B than in Group A.

In cases progressing favourably the amount of work done is increased, and at the end of about three weeks the test exercise produces a greater rise in pulse-rate, say twelve to sixteen beats. By this time the patient is fit to start walking, and has reached Stage 4.

*Stage 4.*—(This corresponds to Scheme 4 in the Appendix.) During the first week or two of this stage the patient only walks along the level, and for a day or two a few yards is sufficient, for although the heart may be capable of the work necessary for a longer walk, the leg muscles need to get accustomed to supporting the weight of the body. (This “flabbiness” of the leg muscles is, however, much less obvious in patients who have had some preliminary leg exercise before getting up.) The duration and pace of the walk are increased until he walks for three minutes at a time, at a rate sufficient to raise his pulse about sixteen to twenty beats. Stair climbing or some similar exercise, such as stepping on and off a stool, is now added. From this stage onwards a rather different method is used in judging the effect of exertion on the heart, namely, that of the pulse ratio.

The meaning of the term pulse ratio was described in an earlier number of the *Reports* (1). Briefly, it is as follows: the pulse-rate is taken with the subject at rest, and then counted continuously for two minutes directly after he has done an exercise lasting three minutes. The total number of beats during the two minutes after exercise, divided by the number of beats per minute counted before exercise, gives the pulse ratio. An example will make this clearer.

Pulse-rate before exercise . . . . .	64
Pulse-rate during the first minute after exercise	84
Pulse-rate during the second minute after exercise	65
Total	149

$$\text{Pulse ratio } 149 \div 64 = 2.33.$$

It was shown (1) that in the same individual the harder the exercise the higher is the pulse ratio; (2) that if two men do the same exercise the one who is physically fitter will have the lower pulse ratio; (3) that in order to obtain the same pulse ratio

in two men the fitter of the two must do a harder exercise than the other. It was also shown that the pulse ratio after moderately easy exercise gives a fairly accurate measure of a patient's efficiency, if the following method is adopted. The patient steps on and off a stool for three minutes at a rate which produces a pulse ratio of 2·5. It was found that a healthy well-trained athlete had a pulse ratio of 2·5 after stepping at a rate of 34·6 times per minute. By comparing this with the rate at which the patient steps, the relative efficiencies of the two can be calculated approximately. Thus if a rate of 6 steps per minute produces a pulse ratio of 2·5 in the patient, his efficiency is  $6 \div 34\cdot6$ , or 17·6 per cent. that of the athlete.

When the patient first starts stepping exercises, the rate should be sufficiently fast to produce a pulse ratio of 2·2—2·3; \* as a rule in case of organic heart disease it is inadvisable for a patient to do any exercise *regularly* which produces a pulse ratio of 2·5, but such an exercise is done occasionally (say once in three weeks) during the period of treatment, for the estimation of his efficiency.

The following case illustrates the use of this method.

The patient was a man of 36, with well-marked mitral stenosis, but no enlargement of the heart. At the time he came under treatment his efficiency was tested, and it was found that a test exercise consisting of stepping on and off a stool six times per minute for three minutes produced a pulse ratio of 2·5; this corresponded to an efficiency of 17·6 per cent. At the end of six weeks' treatment a test exercise consisting of 14 steps per minute produced a pulse ratio of 2·5, corresponding to an efficiency of 40·5 per cent. His efficiency was thus more than doubled as a result of treatment. The table below shows the two exercises used to estimate his efficiency, and the pulse ratios corresponding to the "training exercises"; the latter were such as to produce a pulse ratio of about 2·3, and had to be increased

Date.	Exercise = No. of steps per minute.	Pulse ratio.
Sept. 9	6 steps per minute	2·5 †
" 9	5 " "	2·3
" 12	6 " "	2·3
" 16	8 " "	2·4
" 19	10 " "	2·3
" 22	11 " "	2·3
" 24	13 " "	2·4
" 30	12 " "	2·3
Oct. 6	13 " "	2·4
" 7	14 " "	2·5 †

\* This would produce an immediate rise of pulse-rate of about 20 beats.

† Exercises for estimating efficiency.

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to keep the pulse ratio at approximately this value. This case affords good evidence of the value of progressive muscular work, for according to the man's own account his condition had been stationary for some months; he was not in hospital, and no treatment was given during the six weeks other than graduated exercises.

This scheme of treatment has been found to suit most cases of rheumatic heart disease very well. It may require modification under certain conditions, particularly in patients whose pulse-rate at rest is high (90 or over); in such cases exercises which produce a pulse ratio of 2·3 or 2·4 are often too severe, and a lower pulse ratio (2·1–2·3) should be aimed at.

Exercises producing a low pulse ratio are more suitable in elderly subjects with myocardial degeneration; indeed in such patients it is advisable at all stages of treatment to give exercises which produce rather less alteration in pulse-rate than that recommended in cases of chronic endocarditis.

It may be urged that too much reliance is placed on the behaviour of the pulse as a guide to treatment, and too little on the effect of exercise on the respiration and general condition of the patient. The answer to this is that in Group A the exercises should not cause any *obvious* change in the breathing. It is true that in Group B the exercises ought not to cause any dyspnœa; if the rules given above are followed the depth of respiration is increased, and the rate may be slightly quickened, but there will be no danger of the patient becoming in the least breathless. However we may define dyspnœa, the term does imply some degree of distress; I have never seen any distress produced by an exercise causing a pulse ratio of less than 2·5, provided that the pulse-rate at rest is under 100; as a rule the subject only becomes breathless when the pulse ratio is approaching 2·8–3·0. There is a further advantage in regulating the amount of exercise by the pulse-rate, for it affords an indication that is easy to appreciate; considerable experience is needed before an observer can tell merely by the look of a patient that he has done a little more muscular work than is good for him; if the masseur has not got this experience, other and more easily recognised signs are needed.

It is difficult to assess accurately the therapeutic value of progressive exercises in the two groups.

In Group A the improvement that is noticed may be due only to the recovery in function associated with a decline in inflammation, but treatment on these lines is useful in two ways. Firstly, it avoids any sudden increase in the work of the heart, such as occurs when a patient walks for the first time without

any preliminary exercise in bed; and although tachycardia or slight pyrexia occasionally makes it necessary for a patient to return to bed after being up for a few days, this occurs much less frequently in patients who have had graduated training. Secondly, observations of the effect on the heart of a gradual increase in work afford a daily guide in detecting alterations in the exercise tolerance; if this is deteriorating we have the earliest indication of a recrudescence of the inflammation, and can treat this at the first possible moment.

In Group B there is clear evidence that progressive muscular work increases a patient's capacity for exertion. The need for training a healthy athlete has of course long been recognised, and there is general agreement that it is of benefit to a patient with a diseased heart. The case described and others where figures on the pulse ratio could be quoted show this clearly. There is no doubt that further experience is needed before the best rate of progress is known, but the system described produces a definite increase in exercise tolerance, and at the same time gives a rough measure of the degree of improvement. There is a very real need of the latter, for there is a tendency to rely far too much on a patient's statements in judging of his improvement or deterioration; some patients are good witnesses, but the history of others, however carefully taken, is apt to lead us astray; if exercise tolerance tests are done as a matter of routine, it is surprising how often we find that a patient exaggerates or minimises the discomfort that exertion entails. An example of this was afforded by a woman of 23 with mitral stenosis, and moderate cardiac enlargement. When questioned as to the effect of exercise on her breathing she answered intelligently, and said that it was only on going upstairs that she felt any dyspnœa. She was asked to walk sixty yards along the level at her usual pace, and returned panting, with a respiration rate of 44 and a pulse of 150. On being asked if the walk had made her short of breath, she denied it.

### *Group C. Effort Syndrome*

This comprises a group of patients suffering from dyspnœa, palpitation and pain over the heart, in whom no organic disease can be found. Various terms have been applied to the condition, such as functional heart disease, disordered action of the heart (the D.A.H. of the army), cardiac neurasthenia, irritable heart, etc. None of these terms is free from objection, as they all imply that the seat of the disturbance lies in the heart itself. Lewis calls the condition the "Effort Syndrome," and this

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satisfactorily describes the clinical condition, since the symptoms are brought on or exaggerated by exertion.

This group differs fundamentally from the two former in that over-exertion rarely, if ever, does the patient any permanent harm; care has to be taken, however, in grading the work, for if it is too hard, or on the other hand does not produce a sufficient effect on the circulation, progress will be slow.

It is unnecessary to describe any scheme of exercises to be done in bed, for complete rest in bed does more harm than good, and in all but the worst cases the patient should be up for the whole or at any rate the greater part of the day. During the first few days it is advisable to make the exercises well within the patient's capacity, as he is often excitable and may get attacks of trembling and vertigo, which are due more to the fear of the possible results of unwonted effort than to any direct effect of exertion on the circulation. As soon as confidence is established, harder work is given.

In deciding what effect on the pulse ratio the test exercise should have, we must be guided largely by the pulse-rate at rest. The table given below indicates roughly what pulse ratio the hardest exercise should produce in patients with different pulse-rates.\*

Pulse-rate at rest.	Pulse-rate after hardest exercise.
80 or less.	2·6-2·7
120 or less.	2·2-2·3
120 or less.	2·2-2·3

The pulse directly after exercise in each case will be 140-150.

Stepping on and off a stool is a particularly suitable exercise in these patients, as it is easy in this way to demonstrate to the patient himself that improvement is taking place, a very important matter in those in whom functional nervous disturbance is prominent; patients find that as time goes on the rate at which they can do the stepping exercises is increasing, and the mental effect is excellent. Massage and passive movements are unnecessary in these cases, and it is doubtful whether breathing exercises do any good, even in patients whose respiration is shallow.

It has been said that these patients improve most rapidly if they are allowed to graduate their exercises themselves,

\* Sometimes the patient's pulse-rate at rest falls after some days' treatment; in such cases he will be able to do exercises producing a higher pulse ratio than before.

particularly if this exercise has some objective, as in the case of a game of golf. This is perfectly true in some cases, but a great many need supervision to make them take exercise sufficient to get the best results; unless they are encouraged, particularly in the early stages, many become pessimistic, and this can only be avoided by persuading them to persevere in spite of a slight amount of discomfort.

No account of the treatment of heart disease by massage and exercises would be complete without some reference to the so-called "local heart treatment." This consists of various kinds of massage over the precordial area, and it is supposed in some obscure way to influence the heart muscle, or even the valves. In the older text-books it loomed large, and many beneficial results were claimed for it. Even in a recent publication we read the remarkable statement that it "stimulates the heart to stronger contractions *without increasing its work.*" Lately, however, in most London schools at any rate, less attention is paid to it, and a text-book of physiotherapy published in 1924 omits it completely. Before condemning it altogether, it seemed reasonable to try its effects; careful observations showed that in many cases it produced no alteration in the pulse-rate, and that patients in whom the pulse-rate was slowed (the alteration was only very slight) were of a neurotic type; moreover, in these the slowing could be produced by massage over the right side of the chest, over the forehead, or indeed any part of the body, provided that a general soothing effect was obtained. Local heart treatment is quite useless in organic heart disease; in cases of effort syndrome it is definitely harmful, as it merely serves to direct the patient's attention to his heart, and to foster a belief that there is some cardiac mischief, a belief that it is of the utmost importance to dispel. It is to be hoped that this pernicious practice will soon disappear completely; local heart treatment, together with unscientific teaching, such as that about the danger of raising the arms above the horizontal, can only bring the physiotherapy of heart disease into disrepute. This would be most regrettable, for treatment by progressive muscular work is of the greatest value in diseases of the circulation, and rests on sound physiological principles. If, however, it is to take the high place it deserves as a therapeutic measure, it must be purged of theories and practice that will not stand the test of clinical and scientific investigation.

In conclusion, I must express my great indebtedness to Miss Angove, Sister-in-charge of the Massage Department at Guy's Hospital. The treatment advocated is the outcome of



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our joint experience during the last twelve years, and her help in elaborating it has been invaluable. My thanks are also due to her for writing the Appendix to this paper.

### REFERENCE

- <sup>1</sup> Hambley, Hunt, Parker, Pembury, and Warner: *Guy's Hosp. Rep.*, lxxii. 367, 1922.

## APPENDIX

### A SCHEME OF TREATMENT FOR THE VARIOUS STAGES OF HEART DISEASE

#### *Scheme 1*

1. Lying, deep breathing (*a*).
2. Lying, leg kneading, followed by foot bending and stretching (6 times).
3. Lying, deep breathing.
4. Lying, arm kneading, followed by wrist bending and stretching.
5. Lying, deep breathing.

#### *Scheme 2*

(End of two or three weeks.)

1. Lying, deep breathing.
  2. Half lying, (*b*) leg outstretching (10 times). (*c*)
  3. Lying, head backward bending (6 times). (*d*)
  4. Sitting, alternate trunk rotation (6 times). (*e*)
  5. Lying, deep breathing.
  6. Half lying, double arm bending and stretching (10 times a minute, for three minutes). (*f*)
  7. Lying, deep breathing.
- No. 6 is the test exercise.

#### *Scheme 3*

1. Lying, deep breathing.
  2. Half lying, double leg bending and stretching (10 times). (*g*)
  3. Wing sitting, trunk backward bending (6 times). (*h*)
  4. Wing sitting, alternate trunk rotation (8 or 10 times).
  5. Sitting, two-arm bending and stretching (1 lb. weights 20 times).
  6. Sitting, knee bending and stretching (10 to 20 times, alternate knees). (*i*)
  7. Lying, deep breathing.
- Nos. 2 and 6 are the test exercises.

Here the test exercise is continued for a period of three minutes. At this stage the patient should be ready to get out of bed.

*Scheme 4*

1. Sitting, double arm raising sideways (6 times). (*k*)
2. Walking, (ten yards) at the patient's own pace.
3. Lying, breathing.
4. Wing sitting, trunk backward bending (6 to 8 times).
5. Wing sitting, alternate trunk rotation (quickly). (*l*)
6. Rest.
7. Sitting, double arm bending and stretching (2 lb. weight).
8. Wing standing, heel raising and knee bending. (*m*)
9. Sitting, two-arm rotation out. (*n*)

No. 7 is the test exercise.

When walking lasts for three minutes it forms a test exercise.

*This scheme is progressed by—*

- (a) Increasing walking (distance, time and rate).
- (b) Stepping on and off a stool 13 inches high, for a period of three minutes (at the rate of 6 times a minute to begin with).
- (c) Increasing the vigour of all other exercises or adding to the whole scheme above.

The above is a series of schemes for the treatment of the various groups.

Group A. *Acute carditis*.—Begin with the first scheme, and progress according to chart.

Group B. *Chronic cardiac insufficiency*.—Begin with the first scheme, and abdominal massage.

Group C. *Effort Syndrome*.—Begin with the third scheme, quickly progress to the fourth, and on to a much harder course of educational gymnastics, avoiding exercises which fix the chest wall and so impede respiration.

*Explanation of Terminology used in the Scheme*

(a) *Lying*. Patient lies on back with pillow under head, and does deep breathing.

(b) *Half lying*. Lying with three or four pillows.

(c) *Leg outstretching*. Patient stretches the leg out from the fully flexed position by moving hip or knee.

(d) *Bending the head backwards*, against the resistance of an operator.

(e) Patient sits with hands on hips and turns the trunk from side to side.

(f) Patient stretches the two arms up above his head, and then right down to the side.

(g) *Double leg bending and stretching*. Patient bends his legs up to the trunk, and then stretches them out till they rest on the bed.

(h) The patient sits with hands on hips and pushes the trunk backwards against the resistance of an operator.

(i) The patient sits on a stool or the edge of the bed and bends and stretches his leg.

(k) The patient sits on a stool and raises his two arms to the horizontal and down again.

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(l) The patient sits on a stool with hands on hips, and turns trunk to right and left alternately.

(m) The patient stands with hands on hip, raises himself on his toes and lowers the body by bending hip and knee, and then rises again.

(n) The patient sits with hands to side and turns arm in and out.

DAILY PROGRESSION.—Scale to guide progress.

Day.	Wrist bending and stretching.	Ankle bending and stretching.	Elbow bending and stretching.	Knee bending and stretching.	Double arm bending and stretching.	Leg out- stretching.	Trunk rotation.
2	7 times	6 times					
4	8 "	7 "					
5	9 "	8 "	4 times				
6	10 "	9 "	4 "				
7	11 "	10 "	5 "				
8	12 "	11 "	5 "	4 times			
9	13 "	12 "	6 "	4 "			
10	13 "	13 "	7 "	5 "			
11	8 "	8 "	8 "	5 "			
12	8 "	8 "	9 "	6 "	7 times		
13		8 "	9 "	7 "	8 "	6 times	
14			10 "	8 "	8 "	7 "	4 times

# SOME CASES OF ULCERATIVE COLITIS

## I

By CYRUS IVE, M.B.

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DURING the six months in which I was house-physician to Dr. A. F. Hurst and Dr. G. H. Hunt, I was able to observe closely six cases of ulcerative colitis. I had the good fortune to see five of these cases shortly after the onset of their symptoms, to watch their progress, and to be able to discharge them in excellent health. The sixth case was a legacy, much deteriorated in health and handed down through successive residents over a period of nearly two years, and I should have been satisfied in leaving it as such to my successor. As a matter of fact this legacy, inspired perhaps by the rapid upward progress of the other cases of colitis, improved so much that he also was discharged quite fit. Naturally my chief interest is in the treatment and progress of these cases, and it is mainly from that point of view that this paper is written. But I think it is worth while to mention a few of the outstanding points in the etiology, in the symptomatology and in the investigation and diagnosis of these cases.\*

### *Etiology*

Five of the six cases occurred in men. The maximum age was 52 years, the minimum 19 years, and the remainder between 30 and 40 years. One man was a sewerman, and worked under particularly unpleasant conditions: another man made what was for him an unusually severe physical effort, and was at the same time exposed to severe hardship forty-eight hours before the onset of his symptoms. The woman had been feeling rather weak and easily fatigued for some months before her illness, but she had been able to do her work. The remainder could give no reason at all for their misfortune. Not one of the patients had been abroad, or had come into particular contact with people from abroad. Four of them had never experienced a severe illness before: two of them had had similar but milder attacks of colitis some years before.

\* An account of the disease, by Dr. A. F. Hurst, with references, will be found in the *Guy's Hospital Reports*, lxxi. 26, 1921, and in more detail in *Medical Essays and Addresses*, published in October 1924.

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### *Bacteriology*

No constant bacteriological findings were made. In no case were any of the dysenteric group of bacilli isolated. Agglutination tests against the dysentery group were not made. The Widal reaction for typhoid and paratyphoid fever was negative in all cases. The *Entamoeba histolytica* or its cysts were not found.

### *Symptoms*

Five of the cases had an almost identical onset, and gave a similar history, the variations depending upon the severity of the attack and upon the mental make-up of the patient. The sixth case differed in some respects, and I will give the history in detail later.

The diarrhœa usually started with three or four motions a day, and increased in a few days to any number between six and twenty. In every case discomfort in the hypogastrium and flatulence was complained of; tenesmus was also present as a rule. Rapid and progressive weakness was felt, and loss of weight noted. Fever was present in all cases, the chart showing a swing between 99 and 101° F.

The woman in the series adds a little variation to the story, for in addition to the symptoms described above, one of her first and chief complaints was pain in the right hip, knee and second toe. This pain was due to well-marked polyarthritis.

### *Diagnosis*

The symptoms, negative physical examination, and naked-eye inspection of the fæces pointed to the diagnosis. In all cases the diagnosis was confirmed, and then the progress of the case watched, by means of the sigmoidoscope. The fæces had a very definite appearance: they were liquid, and coloured red by the intimate mixture of blood with them; here and there were to be seen small yellow points of pus, with larger, greyish lumps of mucus. Undigested food was sometimes noticed, especially if the diet was not strict.

Microscopical and chemical tests on the fæces are interesting, but not as a rule helpful or necessary at this stage of the disease.

### *Sigmoidoscopy*

In cases of ulcerative colitis no previous preparation is necessary, but all treatment by colonic lavage should be stopped three or four days before the examination is made. It is of the utmost importance to see the mucous membrane of the colon in its natural state, whether normal or diseased. One colon

douche with isotonic saline solution is sufficient to produce hyperæmia, and if silver preparations or tannic acid have been used, coagulation of the muco-pus and the surface of the ulcers may occur and so mask the true picture. An anæsthetic should never be given for sigmoidoscopy. It is unnecessary, as the only pain caused is due to the stretching of the sphincter ani, and this can be reduced to a minimum if the instrument is inserted steadily and if no force is used. Moreover, it is impossible to place an anæsthetised patient in the most advantageous position for carrying out the examination. In fact, anæsthesia for sigmoidoscopy introduces an element of danger into the examination, for if by faulty manipulation damage is being done to the gut, there is no danger signal from the patient. Excessive pain is usually due to associated conditions such as fistula in ano, and a cocaine suppository or sempule inserted half an hour before the examination will be sufficient to prevent this. The position of the patient during sigmoidoscopy is of great importance. He should be made to kneel upon the table with his feet just over the end: he should then bend the body forwards until his head is resting on a pillow, with his elbows resting on the table. The thighs should be perpendicular and the back hollowed out. The instrument should be inserted by gentle pressure in a downward direction at an angle of  $45^{\circ}$  with the horizontal. As soon as the sphincter has been dilated and passed, the hand should be depressed until the line of the sigmoidoscope is pointing towards the sacral promontory. The instrument slips in with the greatest ease for a distance of 4 to  $4\frac{1}{2}$  inches, when slight resistance will be felt. It is now necessary to insert the lamp and progress by vision. The lumen at the pelvi-rectal junction may be difficult to find, as here the bowel makes an acute turn, generally to the right, and the opening may be missed. By withdrawing a few cms. the opening as a rule becomes obvious, and the instrument may be passed on for a distance of 10 to 14 inches if necessary.

### *Treatment*

The cases I am describing respond in different ways to different forms of treatment. In some the course of the improvement was obvious; in others it was obscure. I will analyse later each case, its treatment, and the result of treatment. But I first want to mention the immediate treatment given to all the patients to relieve their discomfort, and to prevent aggravation of their symptoms.

(a) *Diet*.—The principle is to give an ample diet, but one which provides a minimum of fæcal residue. It consists chiefly

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of boiled eggs, boiled fish, thin bread-and-butter without crust, mashed potatoes, jellies, custard and milk. Green vegetables, badly cooked potatoes, and spiced foods must be avoided. Fruit juice can be given, but no whole fruit should be allowed. Such a diet will often reduce considerably the number of motions passed *per diem*, and will certainly reduce the amount of abdominal discomfort.

(b) *Magnesium sulphate*.—Although most of the motions passed are fluid, small, hard, scybalous masses may sometimes be seen. Until these scybala are got rid of, the patient often has severe colicky pains. A drachm of magnesium sulphate given every morning fasting will prevent the formation of the constipated masses, relieves the patient a great deal, and incidentally assists in lavage of the bowel.

(c) *Charcoal*.—Most of the patients complained of severe flatulence following food. Half-an-ounce of animal charcoal made into an emulsion with milk, sweetened with sugar, and given half-an-hour after meals will prevent this. If the patient is unable to take the ordinary powdered charcoal, two drachms of Fraudin's charcoal pellets may be swallowed instead.

The other forms of treatment depend upon the condition and progress of the case.

(a) *Serum*.—Four of the six cases were given the polyvalent anti-dysenteric serum of the Lister Institute. The serum was given intravenously on five successive days, the initial dose being 20 c.c., successive doses being increased until a final dose of 100 c.c. was reached. A subcutaneous dose of  $\frac{1}{2}$  c.c. should first be given, and if there is a marked local reaction, desensitising doses should be given. Of the four cases treated with serum, two very definitely improved, but the results obtained were less constant and less dramatic than in Dr. Hurst's previous series of cases. No severe reactions occurred after any of the injections.

(b) *Colon douches*.—These were given in all cases. In those cases treated with serum, however, the douches were not given until the effect of the serum treatment had been noted by sigmoidoscopic examination. In those cases showing active ulceration solutions of neo-protosil were used; the initial strength used was  $\frac{1}{2}$  per cent. and the strength was then increased gradually to 5 per cent. In those cases in which active ulceration had ceased, but in which hyperæmia of the mucous membrane persisted, tannic acid was used. The strength of the tannic acid solution was one grain to one ounce of water. The douches were given every morning, if possible after a natural action of the bowels, through a rubber catheter, size No. 8, inserted

just within the rectum; the solution was run in slowly under a low head of pressure (equivalent to 12 in. of water), and the patient told to retain it as long as possible. Most patients were able to retain the fluid without discomfort for twenty to thirty minutes. During the injection the patient is put in the "knee-elbow" position. A pint to a pint and a half is the usual amount used.

(c) *Blood transfusion*.—This was tried as a last resource in two cases. In one it had no obvious effect; in the second its effect was dramatic, and the patient made a complete and rapid recovery.

#### *After-treatment*

This is most important, and a relapse may be due entirely to neglect in following out instructions. The diet must always be regulated, and for a time should approximate to that described above. Later chicken and meat may be added. Tough meat, porridge and wholemeal brown bread should be prohibited, and all vegetables should be passed through a sieve. The teeth should be carefully examined, and dental sepsis dealt with to prevent secondary infection of the colon. A good masticating surface must also be provided. Liquid paraffin should be taken regularly throughout life, to avoid mechanical irritation of the mucous membrane of the colon by hard scybala.

#### *Analysis of Cases*

*Case 1*.—Male, aged 52 years. A moderately severe case with five to six motions *per diem* with pain made worse by the passage of scybala, and with considerable loss of weight, and anæmia. Hb. 64 per cent.; temp. up to 99° F.

Sigmoidoscopy: the mucous membrane was very inflamed, and there were numerous small superficial ulcers with irregular edges and sloughing bases; occasional polypoid outgrowths were seen.

*Treatment*.—Great benefit from magnesium sulphate and charcoal. Full course of serum given, with immediate subsidence of fever. Second sigmoidoscopy two weeks after course of serum showed colonic mucous membrane hyperæmic: no active ulcers to be seen, but some healing ulcers still exuding pus. Tannic acid douches ordered. Sigmoidoscopy three weeks later showed marked papillomatous outgrowth of mucous membrane at pelvi-rectal flexure. This appearance was so marked that it was thought a mistake in diagnosis had been made; that the symptoms might be due to cancer with secondary ulcerative colitis below, and that the improvement in the patient's general condition might be due to the local treatment. A laparotomy was therefore performed. At the operation there was no evidence of growth, but there was thickening of



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the wall along the whole length of the gut, with some adhesions and pericolicitis. The patient's condition continued to improve; his weight increased by eight pounds in a few weeks: the hæmoglobin rose from 62 to 80 per cent., and the diarrhœa disappeared. All the teeth were removed because of the septic condition of mouth. Final sigmoidoscopy: slight papillomatous condition of the colon still present. Healed ulcers seen. No active ulceration present.

This patient was discharged fit after three months in hospital. The papillomatous condition seen was probably an exaggeration of the normal process of regeneration and repair of the mucous membrane.

*Case 2.*—Male, aged 38 years. A mild case passing four to five blood-stained, fluid stools *per diem*. Swinging temperature between 98° and 101° F. Had a similar attack in 1922, which was treated with injections of horse-serum, anti-streptococcal serum, anti-dysenteric serum, emetine and lavage. He then had very severe serum reactions. He made a good recovery, and was quite fit until this second attack started.

Sigmoidoscopy: the colon is inflamed, red, granular and bleeds easily; several small, superficial and isolated ulcers seen; there are hæmorrhages in the submucous tissue.

*Treatment* by anti-dysenteric serum. Desensitising doses were first given, and no reaction occurred. The temperature at once settled down, and the number of stools diminished. Blood was present in the stools for some time after, but pus disappeared within a few days. Second sigmoidoscopy two weeks after course of serum: mucous membrane hyperæmic and congested, and bleeds easily; no active ulceration. Tannic acid enemata were then given, and the bleeding ceased in a few days. Patient discharged feeling very fit.

*Case 3.*—Male, aged 46 years. Severe attack, passing fifteen to twenty liquid blood-stained stools *per diem*, with occasionally hard lumps of fæces. Severe abdominal pain and tenesmus. Considerable loss of weight. Irregular temperature oscillating between 97° and 101° F.; Hb. 92 per cent.

Sigmoidoscopy: mucous membrane of colon thicker and redder than normal, but comparatively dry; several ulcers seen about the size of a sixpenny-piece, rather deep, with irregular margins and sloughing bases; many smaller, superficial ulcers also seen.

*Treatment.* A course of serum had no effect; pyrexia continued, though not so marked; steady loss of weight; Hb. fell to 77 per cent. Lavage with protosil solution. Sigmoidoscopy seven weeks after first examination: mucous membrane less injected and œdematous; numerous small ulcers still present, covered with muco-purulent exudate; at the edge of some ulcers small sessile polypi are to be seen.

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Tannic acid douches now given. Some very septic teeth were removed. Patient gradually improved, and was discharged fit.

In this case it was impossible to say what part of the treatment brought about the improvement.

*Case 4.*—Male, aged 34 years. Severe attack, passing eighteen to twenty motions *per diem*. Had a similar but milder attack in 1916. Severe pain in hypogastrium and flatulence. Very anæmic, with Hb. 60 per cent.; weak; considerable loss of weight. Oscillating temperature between 98 and 101° F.

Sigmoidoscopy: many small, rather deep ulcers with

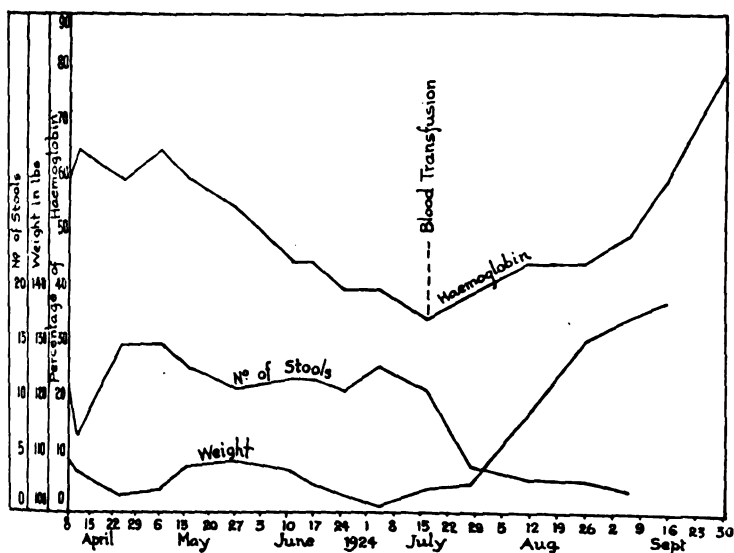


CHART I.

clean-cut edges; some ulcers are situated on red elevations; intervening tissue normal; suggestive of amœbic dysentery.

*Treatment.* Emetine hydrochloride gr. 1 was given intramuscularly for ten days. This had no obvious effect. Sigmoidoscopy three weeks later: mucous membrane still has shaggy appearance; several small, active ulcers seen with sloughing bases; much granulation tissue present. A further course of emetine was prescribed, but with no benefit. Tannic acid douches were then given, but the patient continued to get worse, losing weight rapidly, and the hæmoglobin falling to 35 per cent. A blood transfusion of 500 c.c. was given, and the patient was put on the balcony. There was an immediate all-round improvement. The temperature at once fell and the diarrhœa ceased. The patient put on 2½ stones in weight in two months, and his hæmoglobin percentage rose rapidly. Final sigmoidoscopy: slightly hyperæmic, otherwise

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normal mucous membrane. He was discharged feeling very fit. (See Chart.)

*Case 5.*—Female, aged 27 years. A moderately severe case, passing seven to nine motions *per diem*. She also had an acute arthritis with effusion in the right knee, and acute periostitis of the proximal phalanx of the second toe of the right foot. Her condition was complicated by a fistula in ano. Temperature moving between 99° and 101° F.; Hb. 55 per cent.

Sigmoidoscopy: an annular swelling observed about 6 inches from the anal orifice; the lumen of the gut was here reduced to about 4 mm., but was slightly dilatable by inflation. The mucous membrane was very congested. A clear view was obscured by the constant passage of blood. The presence of the obstruction was confirmed by a second sigmoidoscopy in a surgical ward.

The association with the anal fistula, arthritis and periostitis suggested that the swelling of the colon was tuberculous. A laparotomy was therefore performed. At the operation nothing was found beyond signs of severe colitis and pericolitis of the ascending, transverse and descending colon; the abdomen was at once closed. The obstruction was presumably caused by spasm.

Apart from diet no further treatment was given, yet following the laparotomy the whole condition rapidly cleared up and the patient was discharged quite fit. This case was therefore remarkable for the complicating arthritis, and for the rapid and spontaneous cure. Arthritis associated with bacillary dysentery is not rare: it never becomes suppurative, and generally disappears quickly with rest and improvement in the condition of the colon.

*Case 6.*—Male, aged 21 years. Severe attack in 1922, which cleared up rapidly with serum and lavage. Readmitted to hospital in 1923 with a still worse attack; the relapse was certainly aggravated by the orgy of feasting with which the patient's first discharge from the ward was celebrated. Sigmoidoscopy showed typical ulcerative colitis. During the following year every form of treatment was tried, including lavage through an appendicostomy and blood transfusion. Very little improvement occurred until the patient was put on the balcony in the open air, when he gradually began to recover strength, and was eventually discharged with a completely healed colon mucous membrane. The most interesting feature of his illness was the development of a long stricture of the rectum which was narrowest 5 inches from the anal orifice (Fig. 1). Stricture formation is an extremely rare complication of bacillary dysentery, and Dr. Hurst tells me that he had only seen it occur in one other case of ulcerative colitis or bacillary dysentery (*vide* p. 44). This man is therefore in danger of intestinal obstruction occurring in the future.

He has been warned about this, and to minimise the risk he has been advised to take liquid paraffin and a dose of magnesium sulphate every day to keep his motions soft and thus prevent impaction of faeces with obstruction.



FIG. 1.

Radiogram taken after a barium enema to show narrowing and shortening of rectum and pelvic colon, but normal iliac colon, following recovery from ulcerative colitis.

### *Conclusions*

(a) Serum treatment : two cases were definitely improved, but no dramatic cures were obtained.

(b) Blood transfusion : in one case a dramatic cure was obtained; in the other case in which it was tried a slight rise of Hb. occurred, but was soon followed by a relapse.

(c) Dietetic : colon douches and fresh-air treatment. Under this heading must be placed the two cases which showed no response to other forms of treatment, and which were very slow to improve.

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(d) Spontaneous : one case made a rapid and complete spontaneous recovery.

It is too early yet to say anything about the future of these patients in view of the tendency of previous cases to relapse. At present they are all well, and four of them have been at work for three months. But the risk of relapse is certainly lessened if the advice given under after-treatment is adhered to.

All these cases were in hospital under Dr. Hurst, and I am very grateful for his permission to publish them. I must also thank Sister Addison and her nursing staff for the great patience they have shown in the treatment of these cases.

### II. A CASE OF ULCERATIVE COLITIS: RECOVERY WITH DEVELOPMENT OF A STRICTURE OF THE PELVIC COLON

By J. F. VENABLES, M.D., Assistant Physician, New Lodge Clinic.

THE case under review is of interest firstly in view of recovery taking place after symptoms of such extreme severity had been present at intervals for twenty years, and secondly because recovery was followed by the appearance of a stricture in the pelvic colon, a very rare occurrence after recovery from even the most severe forms of ulcerative colitis. So far as we have been able to ascertain, Case 6 in Dr. Ive's series (p. 42) is the only other case on record of stricture following sporadic ulcerative colitis.

#### HISTORY.

The patient, an unmarried woman of 44, who had never been abroad, first had symptoms of colitis in 1903. For a period of eighteen months both blood and mucus were passed, the stools being slightly loose. There does not appear to have been any constitutional disturbance during this attack; at times the patient experienced slight pain in the left inguinal region. After twelve months medical advice was sought, and the condition responded to treatment after a further six months. Similar attacks occurred in 1905 and 1907. In each attack the general health appeared to be unaffected.

In 1910 the patient passed blood and mucus for three weeks. This attack was abrupt in onset and preceded by sudden very acute pain in the left iliac region. Diarrhoea was continuous throughout the three weeks. The symptoms abated almost immediately on the administration of ipecacuanha.

From 1911 to 1922 she continued at intervals to have bouts of diarrhoea associated with the passage of blood and mucus.

Earlier attacks were relieved by ipecacuanha, but the later attacks did not respond to this treatment. Emetine was tried without success.

For eight months previous to coming under observation the patient had been passing blood and mucus without any symptoms. Three weeks before admission severe pain commenced. At first the pain was situated in both groins, but later it settled down over the ascending and transverse colon. The severe pain only lasted six days, but the diarrhoea, which appeared at the onset of the pain, remained continuous, about fifteen to twenty stools being passed in the twenty-four hours.

The patient was admitted to New Lodge Clinic on September 25, 1923. She was obviously extremely ill and much emaciated. Her temperature varied between  $100.4^{\circ}$  and  $101^{\circ}$  and the pulse rate between 100 and 120. She passed during the first twenty-four hours seventeen stools, which consisted mainly of blood and mucus. Rectal examination, which was intensely painful, revealed an extremely thickened rectal mucosa, in which ulcers could be easily detected with the finger. The patient was too ill for sigmoidoscopy to be carried out.

#### TREATMENT AND PROGRESS.

The administration of anti-dysenteric serum was started on September 27. On that date 40 c.c. were given intravenously. On consecutive days intravenous administration was continued, the dose being increased by 20 c.c. each day until the amount had reached 100 c.c. In all 480 c.c. were given in six doses, 40 c.c., 60 c.c., 80 c.c., and three doses of 100 c.c. There was no dramatic response, but by October 6 the patient's condition had improved sufficiently to admit of examination by sigmoidoscopy. The anal canal was no longer tender. The mucous membrane of the colon was inflamed and ulceration was obviously present. The appearance was described as "grossly abnormal, but suggesting very active healing."

From October 4 to October 13 daily colonic lavage with saline solution was given, and in addition a daily dose of sodium sulphate by the mouth. By October 13 the temperature had fallen to between  $98^{\circ}$  and  $99^{\circ}$ . The number of stools in the twenty-four hours had been reduced from eighteen to ten, and the patient's general condition was much improved. The stool still contained a large amount of blood and mucus. At this point, October 13, tannic acid solution, one grain to the ounce, was substituted for the saline lavage. This was continued daily until October 29, by which date the number of stools in the twenty-four hours was four or five. An organism of the

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Gaertner type, to which the patient's serum showed a marked power of agglutination, having been isolated from the fæces, a vaccine was prepared and its administration was commenced on November 1.

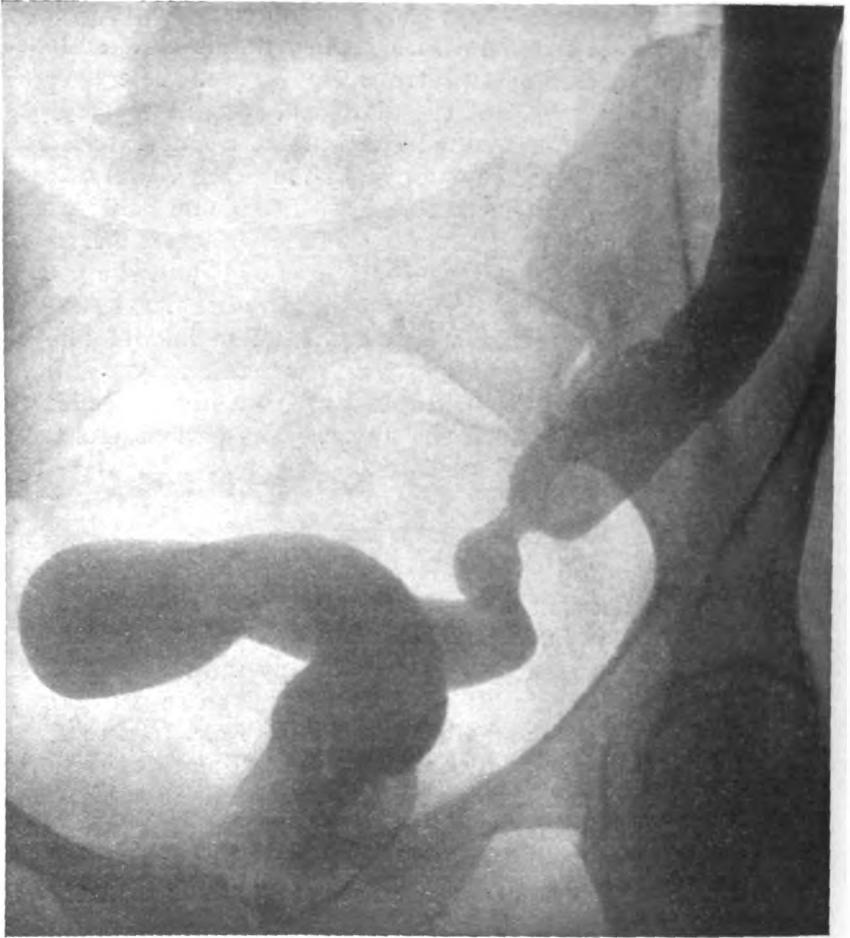


FIG. 2.

Radiogram taken after barium enema showing narrowing of pelvic colon in three places, with rectum and iliac colon unaffected, following recovery from ulcerative colitis.

On November 7 a sigmoidoscopic examination revealed a great improvement. The ulceration was now very superficial and the appearance suggested active healing, the granulating surface of the ulcers being covered with a thin layer of muco-pus. By November 13 the patient's condition had improved immensely; the temperature was constantly normal; the number of stools

varied between one and six in the twenty-four hours, but blood was still present.

On December 4 three teeth showing apical infection were removed.

On December 31 definite islands of pale but healthy mucous



FIG. 3.

Radiogram taken after barium enema, showing normal rectum, pelvic colon and iliac colon for comparison with Figs. 1 and 2 (same scale of reduction).

membrane could be seen on sigmoidoscopic examination, and bleeding occurred much less readily. During December, as the stools became more normal in character, salts and paraffin were given to prevent the faeces becoming solid, so there were still about four or five stools during the twenty-four hours.

In the middle of January the mucous membrane of the colon showed no ulcers, but still bled rather easily. A further examination in February revealed a normal mucous membrane, but,



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whereas at previous examinations it had been possible to pass the instrument the full length, it was now found impossible to pass it more than six inches owing to narrowing and fixity of the pelvic colon at this point. Since the middle of January it had been necessary to give a daily dose of salts and in addition daily lavage, the bowels being open on an average twice in the twenty-four hours with this treatment. During February the stools were kept fairly soft with salts only and the daily lavage was omitted. In view of the obstruction to the passage of the sigmoidoscope a barium enema was given. It was found that the entire pelvic colon was narrowed, less mobile than normal, and apparently shortened. The stricture was irregular, being most severe at three different points (Fig. 2). Fig. 3 is reproduced to show the usual appearance produced by a barium enema in normal individuals.

The patient was discharged completely well in March 1924. She was examined again in July 1924. There had been no recurrence of symptoms. The stools were normal and the sigmoidoscope showed that the mucous membrane was absolutely normal. A barium enema, however, revealed the fact that the narrowing of the pelvic colon had considerably increased, though the stricture had not given rise to any symptoms. She had gained two stone in weight since November 1923. At the latter date she had probably already gained a stone since coming under treatment two months earlier, but she had been too ill to be weighed on admission. The patient is still in good health at the date of writing—December, 1924.

### III. IS MEDICAL OR SURGICAL TREATMENT INDICATED FOR ULCERATIVE COLITIS?

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital.

LOCKHART MUMMERY, in his *Diseases of the Rectum and Colon*,<sup>1</sup> published in 1923, gives statistics of the results of treatment in 82 cases of ulcerative colitis. Of 33 treated medically, 26, or 78 per cent., died; of 49 cases treated surgically, 9, or 18 per cent., died. From these statistics he naturally concluded that all cases of ulcerative colitis should undergo operation, generally appendicostomy, as soon as the diagnosis is made.

His surgical statistics agree closely enough with the only others I have been able to find. Thus Logan<sup>2</sup> collected 51 cases operated upon at the Mayo Clinic, of which 6 died as the immediate result of the operation, and 8 died subsequently. Only 7, however, had sufficiently recovered to allow the stoma

to close within fourteen months. He concludes that "the disease, when recognized, must be considered as most serious. . . . Cure is the exception." Bastedo<sup>3</sup> has recently reported 2 deaths in 5 cases which he had operated upon in New York; the remaining 3 were not benefited by the operation. Strauss<sup>4</sup> of Berlin has had an operation performed in 13 of his cases, 4 of whom died. Adding all the cases of Lockhart Mummery, Logan, Bastedo and Strauss together, we get 118 with 29 deaths, or a mortality of 24·6 per cent. It should be remembered that these do not represent general hospital statistics, but cases operated upon by surgeons specially interested in intestinal surgery.

I am at a loss to know what can be the source of Lockhart Mummery's medical statistics, as they give a totally wrong impression of the results of non-surgical treatment. As his conclusions have been very widely quoted, I have taken this opportunity of summarising the results of treatment of cases which have been under my care since October 1920. Two had previously had an appendicostomy performed, one on my advice, but both were rapidly going downhill when they were admitted under me, and medical treatment was substituted, the stoma being allowed to close.

Since October 1920 there have been thirteen cases of ulcerative colitis admitted under me at Guy's Hospital and at New Lodge Clinic, including the seven reported above by Dr. Ive and Dr. Venables. Not one of these died whilst under treatment, and all went out feeling quite well, passing normal stools, and with a healthy mucous membrane as seen with the sigmoidoscope. Three had relapses after their discharge, but they were of much less severity than the original illness, and the patients rapidly recovered. All were well when we last heard from them, in most cases quite recently.\*

The series includes several cases of great severity, as can be judged from those described in Parts I and II of this note, and from the case recorded in the *Reports* for 1921.<sup>5</sup> The latter, a man of 21, was almost moribund when, in October 1920, he was transferred to my care from a surgical ward, where on my advice an appendicostomy had been performed. No benefit had resulted, and the stoma was not used again, but under medical treatment he recovered completely, and he is still perfectly fit, over four years after his discharge from hospital.

Another case was that of a man of 40, who had had attacks of diarrhoea with hæmorrhage at intervals since 1910, but did not become seriously ill until 1920, when definite ulcerative

\* I have since heard that one patient has recently relapsed.

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colitis was discovered and an appendicostomy was performed. Improvement followed, but two relapses occurred. The second, in April 1922, was very severe, and in spite of daily lavage through the stoma he got steadily worse until he came under my care in October 1922. The usual medical treatment was then instituted, and by December he was well enough to go home, the stools being normal and the mucous membrane as seen with the sigmoidoscope having completely healed. In the two years which have since elapsed he has remained free from intestinal symptoms and has been actively engaged in his profession.

This series of thirteen consecutive cases has therefore no mortality, compared with the mortality of 24·6 per cent. in the collected surgical cases and that of 78 per cent. which Lockhart Mummery has given as the mortality with medical treatment. The conclusion to be drawn is that with adequate medical treatment recovery is almost certain to take place, and that no advantage is gained from surgical interference.

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- <sup>3</sup> W. A. Bastedo: *Med. Journ. and Record*, Sept. 3, 1924.
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## A NOTE AS TO THE IMPORTANCE OF A FINDING OF LEUCOCYTES IN THE "RESTING JUICE" OF THE STOMACH

By ROBERT NORTON GANZ, M.D. Harvard. (From the Medico-Neurological Clinic, Guy's Hospital, and New Lodge Clinic.)

IN view of the occasional finding of large numbers of leucocytes in the "resting juice" \* of the stomach, and in order to investigate the clinical importance of a suggestion by Loeper and Marchal<sup>1</sup> that, since every resting juice contains varying numbers of leucocytes, their examination and estimation should be of diagnostic and prognostic significance as regards the condition of the gastric mucosa, we examined the resting juice of a series of 80 cases in the course of ordinary routine fractional test-meals; controlling our results, at the same time, by examination of the "spittle" of the same patients. In both we counted the leucocytes in fresh specimens with the aid of a drop of iodine solution, noted their ratio to squamous epithelial cells, and further examined the specimens with Leishman's stain.

We found varying, and often large, numbers of leucocytes in every spittle, which rendered unreliable the cell finding, which, also, was often large, in 73 of the 80 resting juices. In two of the remaining seven, the leucocytes, while averaging between 20 and 30 cells per high-power field, were so definitely imbedded in mucus as to suggest an origin from the similarly cell-filled nasal mucus. Of the remaining five cases, four were found to have hypo- or achlorhydria, three being diagnosed as cases of functional dyspepsia, and one being a case of Addison's anæmia. The fifth case was a bleeding gastric ulcer with "high-normal" acidity.

Eight cases of ulcer with hyperchlorhydria were examined. Seven showed less than 10 cells per high-power field, all traceable to the saliva. The eighth case showed between 11 and 20 cells to the high-power field, and there were sufficient cells in the spittle of this case to account for the finding.

\* By "resting juice" is meant the contents of the stomach when fasting before breakfast. By "spittle" is meant the fluid spat from the mouth, and not either sputum from the lungs nor the pure secretion of the salivary glands.

Of thirteen cases of ulcer with high-normal acidity examined, only one showed a cell finding not assignable to extra-gastric origin; this case showed over 30 cells to the high-power field. The diagnosis was bleeding gastric ulcer, and the patient ultimately died. While no autopsy was obtained, there was some suggestion of the possibility of neoplasm.

A second case of bleeding gastric ulcer examined, this one included in the cases with hyperchlorhydria, showed a bile-tinged resting juice and only rare cells.

Eight cases showed hypochlorhydria. Of these four contained less than 10 cells to the high-power field in the resting juice, and a fifth presented between 11 and 20. All of these could be assigned to oral origin or were imbedded in material suggesting nasal mucus. The remaining three cases showed an average of between 20 and 30 cells to the high-power field, while the cell content in the spittle was only between 5 and 10. The ratio of leucocytes to squamous epithelial cells in the resting juice of one case was 30 : 1; in the spittle of that case it was 1 : 1. This difference in ratio was slightly less in the other two cases. The cells in the resting juices were loose and free and gave little evidence of origin from the nasal mucus. There was no evidence of swallowed sputum or free nasal or pharyngeal pus. The diagnosis of one case was rheumatoid arthritis with pyorrhœa alveolaris; the other two cases had been diagnosed "dyspepsia and ptosis."

Eight cases of Addison's (pernicious) anæmia were examined. Five of these showed an average cell content of less than 10, which was exceeded in each case by that of the corresponding saliva. Two cases showed cell contents of between 20 and 30, and the saliva in these cases contained a similar excess of cells. In the eighth case there was a cell content in the resting juice of between 10 and 20, and in the spittle of between 5 and 10, though no other extra-gastric source of cells was evident. There is thus no evidence of inflammatory exudation to support Faber's view that the achylia in Addison's anæmia is always secondary to severe chronic gastritis.

Three cases of gastric carcinoma with hypochlorhydria were examined. They have not been included in this series, as they all had pyloric obstruction and our findings were therefore unreliable. Carcinoma is, of course, the type of case *par excellence* in which pus cells may be found in the stomach.

The following chart includes all the cases examined. The letter "s" following a number indicates the number of cases showing "significant cells" in the resting juice, by which is

# THE "RESTING JUICE" OF THE STOMACH 53

meant only those in which we were unable adequately to trace the cells to extra-gastric origin.

Average no. of cells in resting juice per high-power field.	0-10.		11-20.		21-30.		Over 30.	
	Bile in specimen.	No bile.	Bile.	No bile.	Bile.	No bile.	Bile.	No bile.
Cases of ulcer with hyperchlorhydria . . . . .	3	4	0	1	0	0	0	0
Cases of ulcer with normal high acid . . . . .	6	4	1	1	0	0	0	1s
Other cases—normal high acid . . . . .	7	12	1	2	0	0	0	0
Other cases—normal low acid . . . . .	4	8	0	2	0	3	0	0
Cases with hypochlorhydria . . . . .	2	2	0	1	1s	2s	0	0
Addison's anæmia with achlorhydria . . . . .	2	3	0	1s	2	0	0	0
Other cases with achlorhydria . . . . .	2	2	0	0	0	0	0	0
Total—80 cases . . . . .	61 cases 0s		10 cases 1s		8 cases 3s		1 case 1s	

From this grouping we see some evidence that less than 10 cells per high-power field in the resting juice is almost certainly of salivary origin; between 10 and 20 cells is probably of that origin, while above 20 the source is perhaps worthy of further investigation.

In forty-four cases the mouth was carefully examined for pyorrhœa by Mr. A. Ll. Spencer-Payne, and smears of suggestive material squeezed from around the teeth were stained and examined for pus. The similarity between the degree of pyorrhœa found and the cell content of the saliva is shown in the following chart. It would seem, moreover, that the actual cell finding in the spittle should give a better idea of the amount of swallowed pus than the mere notation of present or absent pyorrhœa.

Average no. of cells in saliva per high-power field.	0-5.	5-10.	10-20.	20-30.	Over 30.
No pyorrhœa (16 cases) . . . . .	14	—	—	2	—
Slight pyorrhœa (5 cases) . . . . .	—	3	—	2	—
Marked pyorrhœa (23 cases) . . . . .	4	5	6	4	4

As regards the source of the leucocytes in the saliva, in health there are present cells from the faucial tonsils, frequently very numerous, which, as described by many histologists, and

here quoted from Jordan,<sup>2</sup> "find their way into the oral cavity, where they are found in large numbers in the saliva as 'salivary corpuscles.'" Any cells from this source are, of course, greatly augmented when pyorrhœa, tonsillitis, or any other inflammatory foci are present.

#### CONCLUSIONS

1. The examination of the resting juice of the stomach for leucocytes without careful control is useless.

2. In a series of eighty cases, seventy-one contained less than 20 leucocytes to the high-power field in the resting juice, and seventy of these counts were rendered valueless by the same or larger numbers of leucocytes being found in the spittle. Of nine resting juices showing over 20 cells to the high-power field, we were unable to assign to extra-gastric origin the findings in four.

3. Of the five cases found to contain "significant cells" in the resting juice, three had hypochlorhydria, another was one of eight cases of Addison's anæmia with achlorhydria, and one a bleeding ulcer with high acidity within normal limits.

4. The series contained no cases of gastric carcinoma, in which pus is undoubtedly often found.

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## THE VALUE OF NEUTRAL RED IN THE ESTIMATION OF GASTRIC FUNCTION

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(From the Medico-Neurological Clinic, Guy's Hospital.)

IN 1908 Field<sup>1</sup> in animal experiments noted that neutral red was eliminated by the stomach. Recently Finklestein<sup>2</sup> confirmed this observation on animals, and showed that congo red, eosin, indigo-carmin, phenolsulphonaphthalein, and other such dyes were not excreted by the stomach.

Saxl and Scherf<sup>3</sup> first suggested investigating the excretion of pigments by the gastric mucous membrane as a method of estimating the secretory activity of the stomach in man. Most observers agree with Simici and Dumitru,<sup>4</sup> that neutral red is the most satisfactory dye to use for this purpose.

Finklestein, Saxl, and Scherf have also shown that the elimination of the dye by the stomach is a function of the glandular secretion.

The present investigation was undertaken to see if by means of this test there would be a simple method of differentiating true achylia, such as is found in Addison's anæmia, in which no gastric juice is secreted, from achlorhydria, in which hydrochloric acid is secreted in the gastric juice but is all combined with inorganic or organic alkaline substances so that free hydrochloric acid is absent from the gastric contents.

### *Technique*

Previous observers have used the intramuscular route for the injection of 5 c.c. of a 1 per cent solution of neutral red in distilled water. The injection is given just as the patient is about to take the test-meal after the resting juice has been withdrawn. All are agreed that under normal conditions the dye then appears in the stomach within half an hour, reaches its maximum concentration in about one to one and a half hours, and disappears within two to six hours.

The excretion of the dye into the stomach results in a pink colour being imparted to the gastric contents in the absence of bile. The regurgitation of bile into the stomach interferes with the test, for neutral red is also excreted in the bile.



As the tint following intramuscular injection of the dye is often very faint, the same dose of the drug was given intravenously in certain cases. Elimination of the dye begins rapidly, reaches its maximum within an hour and ends within two hours under normal conditions. The increased speed of elimination is not accompanied by any obvious increase of intensity of the colour in the gastric contents, so there is no advantage in the intravenous injection of the dye.

The dye excreted into the stomach is obviously greatly diluted by the pint of gruel which constitutes the ordinary test-meal. In a few cases the injection was given as the resting juice was withdrawn and an attempt was made to aspirate any of the dye which might appear within the stomach in the next half-hour. The meal was then given. This delay of half an hour did not seem to be of any advantage.

Amyl alcohol extracts the dye from the acid stomach contents, but any attempt to graph the elimination of the dye by colorimetric comparisons of amyl alcohol extracts of fixed quantities of the different fractions is so inexact as to be useless. The concentration of the dye in the gastric contents is seldom greater than one in one hundred thousand, and usually is less than this.

The range of neutral red is just about neutrality. In no case in the series did the addition of acid to the gastric contents bring out the colour of the dye when it had not been visible previously, but where the total acidity is less than ten it may be advisable to add a little decinormal hydrochloric acid to see if the tint will develop.

### Results

The results of this investigation may be summarised as follows :

(1) In four definite cases of Addison's anæmia (one confirmed *post mortem*) there was no elimination of the dye into the stomach.

(2) In one case of carcinoma of the pylorus and of the stomach with complete achlorhydria, and in a case of ulcerative colitis with a trace of free acid in only one fraction, the dye was not excreted.

(3) Of twelve patients whose test-meal curves were within normal limits, neutral red was recognised in the gastric contents of nine. The remaining three patients (all of whom had been given neutral red intravenously) showed no trace of colour in the gastric contents. One of them was suffering from myxœdema and tabes, while the other two had slight dyspepsia without signs of organic disease.

*Conclusion*

These results, showing the irregular excretion of neutral red into the stomach, suggest that the test is of no value in clinical work. The dye is not excreted in cases of achylia, but it is also not excreted in various other conditions, so that the test is useless in the differential diagnosis between achylia and achlorhydria due to complete neutralisation of the secreted hydrochloric acid.

Our thanks are due to Dr. A. F. Hurst, at whose suggestion the test was made; to Dr. J. H. Ryffel, who kindly placed his laboratory at our disposal; and to Mr. Williamson, who went to a great deal of trouble in preparing the solution used.

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# THE TREATMENT OF ASTHMA AT MONT-DORE

## WITH A NOTE ON THE FOUR OTHER SPAS OF AUVERGNE

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital.

IN the large hall of the bathing establishment at Mont-Dore, among the ancient remains which show that the waters were used by the Romans, is the bust of a soldier. It differs from all others which have survived to the present day in representing a man in a paroxysm of asthma—with shrugged shoulders, distended chest and staring eyes. And this asthmatic Roman soldier bears in his right hand a sphere, reputed to be the emblem of recovered strength, showing that the conquerors of Gaul had already discovered two thousand years ago that Mont-Dore was “*le providence des asthmatiques*,” as it is known in France to the present day.

Sidonius Apollinaris, in the fifth century A.D., was the first writer to describe the use of the waters of Mont-Dore in respiratory disorders, but the modern history of the spa only dates from 1805, when a part of the existing buildings were erected. Since these were modernised and greatly enlarged in 1890, the spa has become increasingly popular, and there are now few asthmatics or chronic bronchitics in France who have not benefited by a visit to Mont-Dore.

In view of the almost complete ignorance which exists in England about Mont-Dore and its treatment in contrast with its remarkable reputation in France, it seemed to me that it might be of interest to record my impressions of the “cure” gained from personal experience this summer. I hope that this may act as a precedent and stimulate others to write about their personal experiences in British and Continental spas.

In a lecture delivered in 1904 the late Professor Landouzy<sup>1</sup> of Paris described the treatment as consisting of four principal parts—climate, water-drinking, inhalation and hot baths, with a number of subsidiary factors used for special cases, and in the following description I shall follow his classification, but I am indebted to Dr. Felix Tardif of Mont-Dore for my personal knowledge of the “cure.”

### 1. *Climate*

Mont-Dore is situated in the centre of France, in the mountains of Auvergne, at a height of 3500 feet above sea-level. It can be reached during the season by a day or night through-train from Paris in nine hours. Although it is impossible to say what climate is likely to suit any particular asthmatic, as each individual is a law unto himself, a very large majority are free from attacks in the Swiss mountain resorts of moderate height, though an altitude of 5000 feet is generally too great. Mont-Dore is thus situated at a height which appears to be specially favourable for asthma, and considering that nearly every visitor is there for asthma, it is remarkable how few appear actually to suffer from it during their stay. It is one of the coldest and rainiest places in France, and even during the season, which only lasts from the middle of May to the middle of September, the weather is more like that of the west coast of Scotland than what one would expect in the centre of France. The good results obtained from the "cure" are all the more remarkable.

### 2. *Drinking the Water*

The twelve springs of Mont-Dore supply every day nearly a million litres of water at a temperature varying between 100° and 117° F. There is nothing very striking about its composition, its chief constituents being sodium, calcium and magnesium bicarbonates, with a little silica, iron and arsenic, and a considerable amount of carbon dioxide. If it really has the remarkable properties which have been ascribed to it, these may be due in part to its slight radio-activity, but still more to that ill-defined property which, like many other natural waters, it is supposed to derive from the fact that it is drunk "living," immediately it emerges from the depths of the earth, and which is lost when it is bottled. Nearly every spa physician believes that many natural waters possess a remarkable property of this kind, and that it is impossible to substitute for them an artificial water containing exactly the same proportion of chemical constituents.

According to Landouzy the Mont-Dore water is partly eliminated by the respiratory mucous membrane, upon which it exercises an "almost specific effect," comparable to that of the balsams used in the treatment of bronchitis; by its sedative and decongestive properties it is said to diminish cough and expectoration, relieve spasm and increase the amplitude of respiration. At the same time it is supposed to stimulate and regulate the nutrition by increasing oxidation, and the excretion of urates is increased, so the "neuro-arthritis diathesis,"

without which many French authors believe that asthma cannot develop, is counteracted.

The favourable effect of Mont-Dore on asthma is indisputable, but I find it difficult to believe that the waters really have any of these specific properties. Although drinking the waters is regarded as an essential part of the treatment, I cannot help feeling very doubtful whether it has really anything but a psychological effect.

### 3. Inhalation

The treatment peculiar to Mont-Dore, which I should personally regard as most important, is the inhalation. Between 6 and 7 a.m. the patient is called and puts on a white hooded jacket of thick woollen material with a pair of combined woollen trousers and socks with rubber shoes or wooden sabots. This quaint costume gives the place a character of its own, which is maintained during the day by the appearance whenever the sun shines of a pair of wet woollen trousers hanging out of nearly every window in every hotel and house in Mont-Dore. The patient then walks to the *établissement*, unless he is asthmatic, in which case two porters in blue over-alls carry him in a picturesque green sedan-chair from his hotel to the part of the building where his treatment is to be carried out. He rids himself of his jacket in an ante-room, and either with his chest bare or in a sleeveless vest he enters the inhalatorium, a large room filled with the vapour of Mont-Dore water at a temperature of about 90° F. He walks round and round for a period which increases from twenty to fifty minutes as the "cure" progresses. The vapour contains traces of iron, arsenic and silica and a considerable quantity of free carbon dioxide. As the patient walks and talks, his respiration is deeper than if he were inhaling whilst sitting down, and it is quite possible that all the constituents of the vapour reach the pulmonary alveoli, but whether they exert any specific effect is, I think, extremely doubtful. The inhalations are said by Landouzy to be "sédatif," "décongestif," "détersif," "dérivatif," "imbibant," "cataplasmant," "tonique," "astringent," "cicatrisant," "anticatarrhal," "antispasmodique" and "résolutif," as well as "stimulant" or "hyperémiant," though these actions would appear to be the exact opposite of the first two I have quoted. I am not clear as to the exact significance of these various adjectives, but the fact remains that the inhalation relieves spasm and loosens bronchial excretion, so that it is expectorated without difficulty. From what I heard too from patients who had visited Mont-Dore

before, the claims that the treatment is also both "curatif" and "préventif" has a good deal of justification.

#### 4. *Hot Baths*

In the late afternoon nearly every patient has a hot foot-bath of running water at a temperature of 122° F. A few sit in water of this temperature up to the waist for an equal period. I suppose that a congested naso-pharyngeal and bronchial mucous membrane might be relieved by drawing blood to other parts of the body in this way, but I think that the remarkable effects on the respiratory system claimed for this part of the treatment must be exaggerated.

#### 5. *Accessory Treatment*

Every patient gargles with some of the warm Mont-Dore water twice a day before drinking his prescribed dose. At the same time some patients give themselves a nasal douche. Whilst in the inhalatorium any patient with pharyngeal or laryngeal catarrh uses a high-pressure spray of the water for his throat for five or ten minutes, a treatment which is, I am sure, extremely effective.

A nasal douche of the gases derived from the Mont-Dore water, consisting of over 99 per cent. of carbon dioxide, for five to fifteen minutes is very commonly employed. It produces a slight stinging sensation, and it certainly seems to have a considerable effect in overcoming chronic catarrh. It is said to be very effective in hay-fever. Though it is claimed that this treatment owes much of its success to the fact that the gases are a natural product, I think that a douche of ordinary carbon dioxide is well worth a trial at home in such cases.

The majority of patients have a warm bath for ten minutes, followed by a hot shower douche given under considerable pressure for two or three minutes immediately before going into the inhalatorium. The douche appeared to me to have a very stimulating action on the respiratory movements, but I preferred having it alone without the preliminary bath.

#### 6. *Psychotherapy*

No French author has called attention to the psychotherapeutic effect of the Mont-Dore treatment. As I have pointed out elsewhere,<sup>2</sup> asthma depends upon a constitutional and often inherited irritability of that part of the vagus nucleus which controls the motor and secretory activity of the bronchi. This irritable centre may be thrown into activity by chemical, reflex and psychical stimuli, none of which is, however, capable

of causing asthma unless the constitutional over-irritability is present. Although chemical stimuli are probably the most important, and reflex stimuli come next in importance, a psychological factor is present to a varying extent in the majority of patients. Quite apart from the direct effect of emotions, expectation is undoubtedly a frequent cause. A patient, who has had an attack under certain circumstances or in certain places, generally expects to have another attack when these circumstances recur or he revisits these places. The acquisition of a more hopeful attitude and the realisation that attacks are not inevitable under particular conditions often exert a very favourable influence on the course of a patient's asthma.

I have already referred to the physical atmosphere of Mont-Dore; its psychological atmosphere—its atmosphere of cure and its common designation as the “*providence des asthmatiques*”—is, I believe, no less important. I learnt during the war how invaluable was an atmosphere of cure in the treatment of the psycho-neuroses of soldiers, and I am convinced that the reputation of Mont-Dore and the stories patients tell each other about its wonderful effect are not the least important factors in improving the health of the majority of its visitors. It is for this reason that I should not like to see any alteration in the routine of the treatment in spite of my criticisms of the statements which have been made, often on most insufficient evidence, regarding its physiological and pharmacological effects. The prescription detailing the ritual of his treatment which is given to the patient on his first visit to his doctor and the modifications made at the consultations which follow on every fourth day are decidedly impressive. Faith in the efficacy of each item of the prescription is most helpful, and if less time were given to treatment than is actually required, there would be even more time for boredom than there already is. Though there are many beautiful walks in the neighbourhood and several good tennis-courts in the town, there is nothing to do when it rains—and it very often does rain—and little to do in the evenings. This is probably a good thing, especially for the overworked, who can be sure of having a complete mental rest at Mont-Dore, a very important matter in view of the aggravating effect in asthma of nervous exhaustion. For this reason, after the morning visit to the inhalatorium, breakfast is taken in bed and is followed by rest and often sleep for one and a half or two hours.

#### *Conclusions*

I have no doubt that the Mont-Dore treatment of asthma is of real value and it deserves to be better known in England

than it has been in the past. Before a patient goes there he ought, however, to undergo the thorough physical examination and biochemical, radiological and bacteriological investigations, which are required in order that the various toxic, infective and reflex factors in the case can be discovered and, so far as is possible, removed. When this has been done a three weeks' visit to Mont-Dore for two, three or four consecutive summers is likely to be very beneficial, especially if it can be combined in the winter with a visit to the Swiss mountains or an equal period of systematic treatment in England.

#### THE OTHER SPAS OF AUVERGNE

Auvergne is famous for its five spas—Mont-Dore for asthma, Châtel-Guyon for the colon, La Bourboule for glands, St. Nectaire for the kidneys, and Royat for the heart and blood vessels.

##### *Châtel-Guyon*

Châtel-Guyon is the most attractive of the five towns, being beautifully situated at a height of 1300 feet above the sea. The waters are hot and gaseous, and are peculiar in containing a large quantity of magnesium chloride—0.156 per cent. more than those of any other known spring. It is to this that their undoubted action on the bowels is due, though when given in properly regulated doses they are not actually aperient. Patients suffering from chronic constipation, muco-membranous colic, the milder forms of non-ulcerative colitis, and the sequels of intestinal infections, such as dysentery and other forms of typical diarrhœa, are generally much benefited by a summer visit of three or four weeks to Châtel-Guyon.

The treatment consists firstly of drinking the waters, the dose of which is carefully regulated so as to help to restore the normal activity of the bowels without producing the slightest irritation or diarrhœa. Baths in running-water at its natural temperature of 94° F., combined with a "sub-marine douche" over the abdomen at a temperature of 113° F. and at a pressure which can be varied according to the case, prove both stimulating for sluggish and atonic bowels and sedative for spasmodic and painful conditions, such as that associated with muco-membranous colic. A mud-poultice, which has been soaked in the water at a temperature of about 115° F., applied to the abdomen either in the bath or apart from it, is also much used for its sedative action.

I was interested to hear from Dr. A. E. E. Reboul<sup>3</sup> that the colon douches, which were as popular before the war in Châtel-Guyon as in Harrogate, where they had also been introduced



from Plombières, have now fallen into disfavour. This is due to the fact that experience has confirmed the criticism of the late Albert Mathieu of Paris, who said that he had seen more cases of colitis caused by the Plombières treatment than cured by it, an observation with which, as I have often stated, I entirely agree. The treatment is now no longer used for mucomembranous colic, for which it was first introduced at Plombières, but is reserved for cases in which it is necessary to remove large accumulations of fæcal matter and for very severe cases of constipation, especially with stasis in the cæcum and ascending colon. For the latter *masso-lavage*, or massage of the bowel when filled with water, is practised two or three times in the course of the "cure." I was very glad to find, too, that the excessive quantity of water and the high pressure formerly in vogue have now been completely abandoned, and that the tube is only passed a short distance into the rectum, as it has at last become recognised that a long tube inserted many inches for the so-called "high enema" simply curls round and rubs the rectal mucous membrane, as I first pointed out sixteen years ago.<sup>4</sup> I am sure that Harrogate would do well to follow the excellent example of Châtel-Guyon, and no longer abuse a method of treatment, the indications for which are extremely limited.

Lastly, a very satisfactory understanding exists between the physicians and hotel-managers of Châtel-Guyon, which results in the provision of a suitable diet for the different types of cases coming under treatment.

The "cure" is thus in many cases similar to that obtainable in Harrogate. It is certainly one to be recommended for patients with chronic intestinal disorders, who wish at the same time for a thorough change of surroundings and warm weather in a place where one can rest assured that the treatment will not be overdone and that the important question of diet will be properly supervised. With Châtel-Guyon available there is certainly no justification at all for sending English patients to the German spas and clinics, which specialise in the treatment of diseases of the bowel.

### *La Bourboule*

The waters of La Bourboule are among the richest in arsenic of any in existence, a litre of the water from the different springs containing a quantity which is equivalent to from 8 to 31 minims of Fowler's solution.<sup>5</sup> Whether the fact that the waters are natural really gives them any advantage over an equivalent quantity of Fowler's solution, as is not unnaturally claimed for them, is, I think, doubtful. But the place is a very pleasant

one, and everything is done in the way of providing suitable exercise and amusement for the large numbers of children who go there every summer. There is no doubt that La Bourboule, which is situated 2880 feet above the sea-level, should prove very useful for children with enlarged glands, as well as for young women suffering from mild grades of anæmia. It is also possible to undergo at La Bourboule similar treatment to that of Mont-Dore, so that the place can be regarded as an alternative to the latter for asthma, especially in children.

### *St. Nectaire*

St. Nectaire is a picturesque little town situated twenty miles from Mont-Dore, from which it can be reached by motor-coach. It has a reputation of being useful for cases of nephritis and urinary infection after the acute stage has been passed. There is nothing very notable about the composition of its alkaline waters or about the treatment, the only special feature of which consists of so-called "lumbar sprinkling."

### *Royat*

The climate of Royat is somewhat enervating and the absence of any level walks seemed to me to take away considerably from its suitability as a place for the treatment of cardiac disorders. The treatment is mainly with carbon dioxide baths and is very similar to that which made Nauheim famous before the war.

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## A CASE OF RHEUMATOID ARTHRITIS: A MEDICAL AUTOBIOGRAPHY

By J. N. A.

J. N. A. is an ex-infantry officer, who served in France from February 1915 to May 1917, when he was wounded, with the exception of four months at home following a slight wound in the first battle of the Somme in July 1915. On the second occasion both wounds were very septic. His first acute attack of "rheumatism" occurred in December 1917, at the age of 23, whilst with his reserve battalion in England. His temperature rose to 103° F. and most of the larger joints were affected in turn. The pain was excruciating in character but relieved by salicylates. He was discharged while under treatment in April 1918, being unfit for further active service.

His second acute attack took place in November 1919 whilst a medical student at Guy's Hospital, starting with a temperature of 101° F. and sore throat; very severe pain and swelling occurred in the large joints in succession. This attack was again benefited by sodium salicylates, and recovery ultimately occurred after tonsillectomy and the administration of autogenous vaccines prepared from streptococci isolated from the tonsils.

His third attack commenced in December 1921 and lasted for six months. It began with equally severe pain and swelling in most of the large joints in succession, while finally the metacarpo-phalangeal and proximal inter-phalangeal joints of the hands were involved.

The teeth were all x-rayed in January 1922, but apart from four dead teeth no other abnormality was detected. In the opinion of high dental authorities no useful purpose could be served by extracting any or all of the teeth. On applying pressure to the gum over many of the teeth, however, white fluid matter was expressed. On account of this, as no other source of infection could be discovered in the nasal sinuses or elsewhere, Dr. Hurst recommended that six of the teeth should be extracted. This was done on February 22, and was followed by

(1) Pyrexia which was maintained for nearly a fortnight at or about 100° F.;

(2) Profuse and persistent secondary hæmorrhage from the teeth sockets;

(3) A severe "flare up" of the metacarpo-phalangeal and inter-phalangeal joints, which became swollen, flexed and acutely painful.

These phenomena all commenced within four days of the extractions, the temperature rising the same evening and the serious hæmorrhage and acute inflammation of the joints three days later.

Salicylates now had little effect, but considerable relief followed fixation of the affected joints. Although the coagulability of the blood was found to be normal, calcium chloride was given intramuscularly and calcium lactate by mouth before the next extractions. On April 7 four more teeth were extracted. These extractions were followed by

(1) Slight pyrexia;

(2) Negligible hæmorrhage;

(3) Severe "flare up" in joints (left knee, left ankle and both hands). On April 21 six more teeth were extracted. This was followed by

(1) Slight pyrexia;

(2) Severe "flare up" in joints (knees, hips, hands, elbows).

On June 9, 1922, an autogenous vaccine obtained from matter expressed from the gums was given. This injection was followed by

(1) Slight pyrexia;

(2) Slight reaction in joints (including sterno-clavicular and sterno-costal).

Seven days later a second dose of vaccine was given with almost negligible reaction.

On June 28, 1922, six teeth were extracted, and on June 30 the last six teeth were extracted. On both these occasions the extractions were followed by only slight pyrexia and no joint pains.

From the commencement of this prolonged attack until these last extractions the patient's weight had fallen from 10 st. 8 lbs. to 8 st. 10 lbs., and he had become markedly anæmic. Thanks to the continuous splinting of affected parts, however, there were no deformities in any of the joints. All active trouble having apparently ceased after the removal of the last teeth, it only remained to regain full movements in all joints, and to improve as far as possible his general condition. This was rapidly effected at New Lodge Clinic, where, after a course of massage and exercises with injections of ferrarin and autogenous (dental) vaccines, in six weeks the patient had regained full movements in all joints and over a stone in weight.

## 68 A CASE OF RHEUMATOID ARTHRITIS

In September 1922 complete upper and lower dentures were fitted, since which time there has been no recurrence, the patient being now (December 1924), two years later, completely fit.

All the teeth showed on extraction exceptionally long fangs, unusually adherent gums, but no obvious abnormality, except that streptococci were grown from one or more teeth of each group which were removed.

The chief points of interest in the case are—(1) the very close simulation of acute rheumatism in the first two attacks; (2) the dental origin of the infection in spite of the almost normal appearance of the teeth, as shown by (*a*) the general and local reaction following extraction and inoculation with vaccines prepared from the teeth, and (*b*) the recovery which only occurred after all the teeth were removed; (3) the complete functional recovery of the joints, due probably to the fixation throughout the acute stage of the illness.

## THE WASTE OF LIFE FROM APPENDICITIS

By R. P. ROWLANDS, M.S., Surgeon to Guy's Hospital.

NEARLY all lives lost from appendicitis are thrown away and wasted, for, in an ideal state, very few deaths should occur from this disease, which is curable by timely operation at a very small risk. In spite of the progress of medicine and surgery the loss of life from this condition steadily increases, and increases faster than the population, so that it would appear to be due to lack of wisdom rather than lack of knowledge.

During 1923 2826 persons died from appendicitis and "typhlitis" in England and Wales <sup>1</sup>—nearly 300 more than in 1913: in the United States of America <sup>2</sup> over 11,000 deaths were recorded in 1920 from the disease. Even so these figures do not tell the full tale, for many additional deaths are returned as due to the complications and sequelæ of this dread disease, and not a few of those who survive suffer severely from these complications and sequelæ. Most people think that appendicitis is well under control, but a study of the results and statistics should wake them up and cure the vanity of all who are directly or indirectly responsible for the public health and education. This waste of life is the more deplorable, because the victims of appendicitis are mostly young people, whose life work has hardly begun—over 50 per cent. being under twenty years of age—and because timely operation would save over 99 per cent.

How can this tragic waste be prevented? Chiefly by better education of the public and the profession, resulting in earlier diagnosis and better treatment. Improvements in the choice of time for operation, in operative technic and in after-treatment should lower the mortality very considerably. Much can be gained and many lives saved by the earlier recognition of the minor attacks, which so often precede the graver ones and should be regarded as warnings of the greater trouble to come. Appendicitis is by far the commonest cause of abdominal pain, "bilious attacks" or indigestion, especially in young people. During these attacks of colic or indigestion the appendix is usually tender, although the temperature may not be raised, and their capricious repetition is always suspicious and generally

conclusive. Every effort must be made to settle the diagnosis, and when the presence of appendical colic, subacute or recurrent appendicitis is decided upon or strongly suspected, the appendix should be removed before a serious or fatal attack develops. In such cases preventive surgery can and should be carried out at a very small risk.

Care must be taken, however, not to remove the appendix indiscriminately, especially in neurasthenic patients, or without exhausting every effort to make a correct and complete diagnosis both before and after the abdomen has been opened. Such indiscriminate, unwise or incomplete operations naturally fail to give good results, and tend to bring discredit upon all operations for appendicitis in the eyes of the public and to encourage further and fatal delay in the proper treatment of the genuine condition.

In acute appendicitis delay is the one great cause of death, and for this the ignorance and apathy of the public are chiefly responsible. The public (and especially those in charge of children) should be taught that severe abdominal pain is a serious symptom demanding the immediate attention of a medical man. The knowledge of such simple and important facts should be broadcasted by the Ministry of Health with the aid of the press, the cinema and the wireless. No medical man can speak with the same authority or without acting unprofessionally, and anonymous contributions carry little weight, but carefully considered and authorised information for the public on such vital subjects is overdue.

At present the doctor is not often called to a case of acute appendicitis until the most favourable opportunity for successful treatment has been allowed to pass, until purgatives and other homely "remedies" have been tried in vain and have done much harm. When he sees such a case, the doctor should do everything possible to arrive at an early diagnosis, and in the meantime put the patient to bed and allow nothing but sips of water to be given by the mouth. Above all he must forbid all purgatives and sedatives. When he suspects acute appendicitis, he has the graver responsibility of deciding without delay if an operation is necessary. To share this responsibility an immediate consultation is imperative, both in his own interest and that of the patient.

The following symptoms and signs are strongly suggestive of acute appendicitis.

Acute pain in the middle of the abdomen, sometimes well above or even to the left of the umbilicus, perhaps settling in the right iliac region. Superficial and deep tenderness and

rigidity in the latter situation or, failing this, lower towards the pelvis or higher towards the loin, where the appendix may lie. The thighs must be flexed and the body supine and at rest while this most important sign is sought. Occasionally the appendix or abscess may be palpable either in the right iliac fossa or in the pelvis from the rectum or vagina. Anorexia, nausea and often vomiting are present in the early or severe stages. The temperature is nearly always raised, the face is frequently flushed, the tongue furred and white, and the pulse is quickened and often bounding. The pain is frequently so intense as to prevent sleep. The bowels are usually constipated, but there may be early diarrhoea, especially when the appendix is in the pelvis. Frequent and painful micturition may also indicate pelvic appendicitis. All these symptoms and signs may abate when the appendix perforates and its internal tension is relieved. This lull in the storm deceives the unwary, but it is often followed in a few hours by signs of spreading peritonitis. Leucocytosis is usually present and is of considerable value in the diagnosis of doubtful cases, especially when a localised deep abscess has developed in an obscure situation, such as high up and far back out of reach of palpation in the hollow of the sacrum or behind the ascending colon.

#### DIAGNOSIS

There are certain pitfalls in the diagnosis of appendicitis, which have to be avoided by careful systematic examination. For some of the diseases which may be mistaken for acute appendicitis an operation is unnecessary and may be harmful. I will therefore discuss these first.

Appendicitis is often mistaken with disastrous results for acute gastro-enteritis arising from any cause, but especially from indiscretions of diet. The latter are often followed by vomiting and gastric pain with diarrhoea. Gastro-enteritis may complicate general infections, such as influenza, but it rarely gives rise to definite localised tenderness with resistance over the appendix. Therefore in any doubtful case this sign must be sought with the greatest care and with the abdominal muscles as relaxed as possible, the thighs being flexed with the body supine and at rest. It is commonly forgotten that the position of the appendix is subject to considerable variation and that deep tenderness is often more important than cutaneous hyper-æsthesia or tenderness over McBurney's point. Fever, usually absent in gastro-enteritis, is nearly always present in the early stages of appendicitis.



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Appendicitis has been mistaken for the cyclic vomiting of young children with dire results, but the local signs of inflammation of the appendix should prevent this serious error. Acidosis is by no means limited to cyclic vomiting, but may follow persistent vomiting and starvation from any cause, including appendicitis.

Appendicitis is also very apt to be mistaken for pyelitis or pyelo-nephritis (especially affecting the right kidney) due to *Bacillus coli* infection or associated with pregnancy or growths of the uterus pressing upon the ureter. Therefore it is of the greatest importance to examine the urine carefully in every case before an operation is advised.

To mistake typhoid fever for appendicitis and *vice versa* is possible on account of the early abdominal pain, fever and constipation commonly present. Moreover, the lymphoid tissue of the appendix is commonly affected in typhoid fever, and definite appendicitis with perforation may develop at any time. Perforation of a typhoid ulcer of the ileum may also simulate acute appendicitis.

Biliary and renal colic are chiefly distinguished from appendicitis by the absence of fever and signs of inflammation and by local tenderness or swelling over the gall-bladder or kidney.

I have known a round-worm cause very severe intestinal colic in an adult, which was mistaken for appendical colic, but the absence of fever and general symptoms make it unlikely that this rare condition should be mistaken for acute appendicitis.

Menstrual colic has been diagnosed in error for appendicitis, but this should be distinguished by the history, the absence of fever, and the pelvic and bilateral distribution of the local tenderness.

Mistaking appendicitis for inflammation of the Fallopian tubes should be carefully avoided, for the latter condition does not often call for an operation, but is better treated conservatively. A careful study of the history and bimanual pelvic examination with the discovery of bilateral swellings and tenderness in the pelvis should prevent this mistake.

Pneumococcal peritonitis is another condition for which an early operation is not often advantageous, but it is sometimes very difficult to distinguish from appendicitis with peritonitis. Sometimes the history, or the presence of pneumonia or pleurisy and the diffuseness of the peritonitis from the beginning, helps us to avoid this mistake.

Appendicitis may be mistaken for early pneumonia or pleurisy on account of the pain and tenderness referred along the lower branches of the intercostal nerves, but rapid respir-

ation, flushed face and abnormal physical signs at the base of the lung soon show themselves in the latter conditions.

Other diseases for which acute appendicitis may be mistaken are not so important as those already described, because they also are best treated by immediate laparotomy. Amongst these may be mentioned peritonitis due to perforation of a duodenal or gastric ulcer, perforation of Meckel's diverticulum or of a diverticulum of the colon, acute suppurating cholecystitis, acute hæmorrhagic pancreatitis and acute intestinal obstruction.

#### TREATMENT

*Early operation.*—Osler,<sup>3</sup> in his standard work on the *Principles and Practice of Medicine*, writes: “Gradually the profession has learned to recognise that appendicitis is a surgical disease. In hospital practice the cases should be admitted directly to the surgical wards. Many lives are lost by temporising. The general practitioner does well to remember—whether his leanings be toward the conservative or the radical methods of treatment—that the surgeon is often called too late, never too early. There is no medicinal treatment of appendicitis. There are medicines which will allay the pain, but there are none capable in any way of controlling the course of the disease. . . . Operation is indicated in all cases of acute inflammatory trouble in the cæcal region, whether tumour is present or not, when the general symptoms are severe, and *when at the end of twelve hours, or even earlier, the features of the case point to a progressive lesion.* The mortality from early operation under these circumstances is very slight.”

Personally, I firmly believe that, unless there is some grave contra-indication to any operation, it is wise to get a good surgeon to operate in every early case *as soon as the diagnosis of acute appendicitis is made or strongly suspected, and the sooner the better*, if possible within twelve hours. This is the best way to avoid troublesome and dangerous complications, and it is to be remembered that appendicitis is dangerous only when the infection is allowed to spread beyond the appendix, and to give rise to serious complications, which must take some time to develop and are therefore preventable. Early operation saves the patient a great deal of pain, misery, time and money, and relieves the medical attendant of much anxiety. That it saves time and money is clear; for if the patient gets over the attack he will have to be away from work again during the deferred operation; in this way the time of disablement and the expense are greatly increased. If suppuration develops

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and an operation becomes absolutely necessary late in the attack in spite of conservative treatment, the patient may be bed-ridden for weeks or even months. On the other hand, early operation abolishes the need of drainage and avoids the risk of fistula, hernia or subsequent intestinal obstruction. In the early stage, especially in the first attack, the operation is nearly always easy, because adhesions are few or soft, and it is nearly, if not quite, as safe as an "interval" operation. But no one can foretell the end of an attack of appendicitis under conservative treatment. No disease is more treacherous, for what may appear to be a mild attack frequently ends in perforative peritonitis or death. No one can say what is going on in the appendix or peritoneum until the abdomen is opened, and it is common to find a tense, gangrenous appendix associated with slight symptoms. The lull that often follows perforation is particularly deceptive, free pus being found sometimes in a supple abdomen, especially in children or in women during pregnancy or after labour. Nothing but early operation can remove the cause or set a limit to the spread infection.

Various authorities give the mortality of appendicitis under medical treatment as from 8 per cent. to 12 per cent. At the London Hospital<sup>4</sup> the mortality of 341 late cases treated expectantly was 3·5 per cent. : in 109 of these the attack did not subside, so that an operation had to be performed during the attack, with seven deaths, or 6·4 per cent., and in 232 cases operation was performed after the inflammation had subsided, with five deaths, or 2·1 per cent. The death-rate can be reduced below 1 per cent. by operating at the earliest possible moment. In a long experience I have not lost a single patient operated upon within twenty-four hours; and, as shown by Mutch,<sup>5</sup> there were no deaths in the operations of many surgeons performed within the same interval at Guy's Hospital during the four years 1906 to 1909. At the London Hospital during the years 1920 to 1923 inclusive there were 221 operations within twenty-four hours of the onset of symptoms, with two deaths, or 0·9 per cent. But there is no need to labour this point, for the experience of surgeons all over the world has conclusively proved that the mortality of early operations for acute appendicitis is very low.

### LATE OPERATION

It must be allowed that, although an operation at the earliest possible moment (within twenty-four hours) is by far the safest and best treatment for acute appendicitis, it is often impossible

to get this done. Poor patients, chiefly from ignorance, do not often ask their medical attendant to see them until they are already seriously ill and have tried their homely remedies, more especially a variety of purgatives and sedatives. A capable surgeon is not always available at short notice, the diagnosis may seem doubtful, or the patient may refuse operation. However, if the advantage of early operation were properly appreciated by the public and by the rank and file of the medical profession, it is certain that there would be less delay than there is at the present time.

In late cases, seen after the most favourable time for operation has been allowed to pass, it is sometimes a difficult matter to decide upon the best plan of treatment. After forty-eight hours it is generally best to try conservative treatment, preferably in a nursing home or hospital, and to keep an hourly record of the pulse and temperature. Immediate operations for spreading peritonitis due to appendicitis have a high mortality, but under conservative treatment limiting adhesions usually form and the resistance of the patient is increased, with the result that deferred operations for the drainage of a localised abscess have a much lower mortality. The patient is placed in the Fowler position and given nothing by the mouth. Saline solution, containing 3 to 5 per cent. glucose, is given by the rectum. Hot fomentations are applied to the abdomen to relieve pain. Neither morphia nor a purgative is given. If the attack subsides, as it does in about two-thirds of the cases, the appendix can be removed when all symptoms and signs have disappeared and the temperature has been normal for a week. If, on the other hand, the pulse and temperature do not subside, and the local condition gets worse or a localised abscess forms or increases in size, an operation has to be undertaken. Love found the mortality of 341 delayed operations to be 3·5 per cent., which compares very favourably with that of 103 operations carried out on the third or fourth day, with over 10 per cent. mortality, or with that of 1677 immediate operations, *including both early and late cases*, which was 5·8 per cent. Moreover, the complications in all the late cases treated expectantly were 6·7 per cent. compared with 11·9 per cent. in the late cases subjected to immediate operation. Deaver and Magoun,<sup>6</sup> in a review of 5488 appendicectomies at the Lankenau Hospital, found that the mortality was reduced to 4·2 per cent. in the years 1915 to 1919 owing to the adoption of the expectant treatment for selected cases seen after thirty-six hours. When all cases were operated on at once (during 1901 to 1905) the mortality was 10·5 per cent.

It is most important to remember that no hard-and-fast rule can be wisely adopted for these late cases, for time is by no means the only factor in this difficult problem. Much depends upon the study of all the facts and circumstances of each individual case. Temporising is notoriously unsafe in children and old people, whose resistance to infection is much below that of the adult in the middle period of life. When the circumstances are unfavourable for constant observation and complete control, such as can be obtained in a hospital or nursing home near the surgeon, it is often safer for the *expert* surgeon to operate when he first sees the patient, for such a favourable opportunity may not occur again. Much depends upon the skill of the individual surgeon, for one who is experienced and expert can complete the operation in a few minutes with the minimum of disturbance of the intestines and peritoneum and the least amount of anæsthetic. Under these circumstances the risks of spreading the infection and lowering the resistance are very slight and are more than balanced by the benefits accruing from the removal of the source and products of the infection. A combination of experience, judgment, skill and speed is priceless in these cases, enabling the surgeon to decide exactly what to do and to do it in the best possible way. On the other hand, a less experienced and less skilful surgeon may be wise to temporise, keeping the patient under careful observation and conservative treatment in the hope that the attack may subside, but holding himself ready to operate at once if the pulse, temperature and pain indicate that the disease is taking an unfavourable course.

#### CONCLUSIONS

1. Deaths from appendicitis are far too numerous and are increasing—about 3000 a year in England and Wales. Most of the victims are young people.

2. Timely removal of the appendix would prevent nearly all these deaths. The mortality of interval operations or of operations within twenty-four hours of the onset of acute appendicitis is under 1 per cent., whereas the mortality of late operation is at least 3·5 per cent. and may be as high as 10·5 per cent. for operations performed on the third or fourth day.

3. In many acute cases coming for treatment after forty-eight hours it is safer under good conditions to try conservative treatment in the hope that the attack may subside, so that

(a) the appendix may be removed with greater safety during the quiescent period, or, failing this,

(b) a localised abscess be opened when the resistance of the patient to the spread of infection has increased.

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## SOME NOTES ON THE SURGICAL ASPECTS OF GALL-STONE DISEASE \*

By W. H. OGILVIE, M.Ch., Senior Demonstrator of Anatomy, Guy's Hospital.

### THE SENSATION OF THE GALL BLADDER IN RELATION TO DIAGNOSIS

THE surgeon usually meets with cases of gall-stone disease as the result of some crisis, mechanical or inflammatory, due to the presence of the stones in the gall bladder. The diagnosis in these cases does not usually present much difficulty. It is also becoming increasingly frequent for surgical intervention to be invoked in the treatment of cases of cholelithiasis in which the stones have not given rise to any such catastrophes, but are considered responsible for a general loss of health in the patient. The diagnosis in such cases has been arrived at by a correlation of the history with the results of clinical, chemical, and radiographic examinations, and in some cases the evidence does not amount to more than a strong presumption. But apart from cases in which he operates for a diagnosis of cholelithiasis, the surgeon is continually meeting with gall stones as a surgical surprise, either when, though they are responsible for the train of symptoms for which operation was undertaken, these symptoms have mimicked those of some other abdominal condition, or when stones are found at a laparotomy on a patient in whom they have apparently given no trouble.

The essential symptoms of gall stones are, to say the least, equivocal. Those which, when present, are of the greatest help in diagnosis are due to some complication to which the stones have given rise. Thus, it is generally recognised that pain referred to the right shoulder is due to irritation of the diaphragmatic peritoneum by an inflamed gall bladder. Local tenderness is an indication of a pericystitis. Jaundice and colic are due to mechanical interference. But signs and symptoms

\* Part of the investigations on which these notes are based were used in a Hunterian lecture on Infections of the Alimentary Tract, which will be published in full elsewhere.

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due to the presence of stones which have not given rise to mechanical or inflammatory complications are inconstant. They are referred in the main to the stomach, being those of a flatulent dyspepsia, and it is questionable whether the gall stones are in such cases the cause of the dyspepsia, or whether a derangement of gastric function is not bound up with the causation of the gall stones.

In the hope of obtaining help in the diagnosis of its ailments, I undertook some observations on the sensation of the gall bladder.

It is necessary for this purpose to obtain access to the gall bladder in a conscious patient. As operations in this region are but rarely performed under local anæsthesia, I had to rely upon cases of cholecystostomy. After drainage it is possible to introduce instruments into the lumen of the gall bladder, but it is important that the instruments shall not touch the sides of the tube as they are introduced, or sensation will thereby be transmitted to the abdominal wall, and interpreted by the somatic nerves. The size of the tube which is generally used for draining the gall bladder renders this difficult, and therefore a tube with an internal diameter of 12 mm. was stitched to the opening in the gall bladder in the cases to be investigated, and the free end of the tube did not project more than 10 mm. into the opening. Investigations were carried out after the tube and its passage were sealed off by adhesions, that is, between four days and a week after the operation. The tube was cut short half an inch from the skin, and after the observations had been carried out, its continuity was re-established by a glass connection.

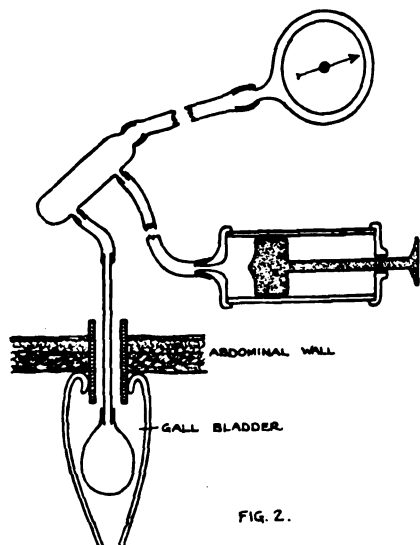
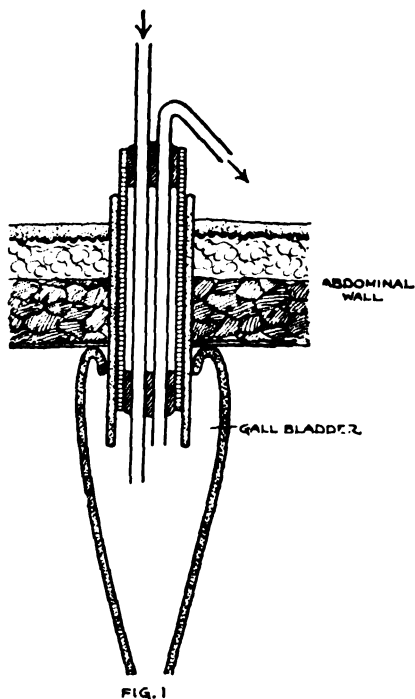
The bile in the gall bladder was first mopped out with small dabs of sterile wool.

Sensation of touch was then investigated, first by passing an aural wool-carrying probe down the tube and stroking the wall of the gall bladder with a wisp of cotton wool, and then by touching the walls more firmly with a naked probe.

To investigate sensation of temperature I first made use of a hollow metal sound, constructed for me by Mr. T. M. Fripp, through which liquid of any desired temperature could be circulated. Owing to the uncertainty as to the position of this sound in the gall bladder, I abandoned it, and circulated a current of sterile saline solution through the gall bladder itself. The apparatus is illustrated in Fig. 1. The saline solution is conveyed to and from the gall bladder by two glass tubes, the entering tube projecting more into the lumen than the return tube. Both pass through a larger glass tube which is plugged at each end with sealing-wax, and which exactly fits the rubber



tube stitched to the gall bladder. When the apparatus is in position, a current of saline solution at any desired temperature may be circulated through the gall bladder, but the warmed or cooled liquid is insulated from the abdominal wall by an air space, as well as by the rubber and glass tubes. Before use, the apparatus was sterilised in alcohol. Saline cooled with ice to about  $40^{\circ}$  F., and warm saline at about  $150^{\circ}$  F., were run through by connecting a Wolff's bottle to the inlet tube, the return tube being connected by rubber piping to another vessel standing on the floor by the bed.



The sensation caused by distension of the gall bladder lumen was investigated by means of the apparatus shown in Fig. 2. A small rubber sound was made by fixing the tip of a glove finger over the end of a fine glass tube. This sound was very small, but could be distended to a diameter of 70 mm. By means of a three-way glass connection, the sound was connected to a 20 c.c. record syringe, and to a manometer. The pressure exerted by its walls was practically constant for all degrees of distension.

Thus, when containing 5 c.c. air, it exerted a pressure of 45 mm. Hg.

"	"	"	10	"	"	"	"	50	"
"	"	"	20	"	"	"	"	40	"
"	"	"	30	"	"	"	"	40	"
"	"	"	40	"	"	"	"	40	"
"	"	"	50	"	"	"	"	40	"

It was therefore easy to arrive at the pressure exerted on the walls of the gall bladder. The sound was introduced till the end of the glass tube projected just beyond the rubber drainage tube, and the bag was entirely within the lumen of the gall bladder. Air was then slowly introduced by means of the record syringe, and sensations noted by the patient were jotted down by an assistant, the readings on the manometer at the time being also recorded. Areas of hyperæsthesia were looked for by stroking the skin of the abdomen and back when pain was recorded by the patient.

Seven cases were investigated with regard to sensations of touch, temperature, and distension. In one of these no sensation was evoked by the sound, the probable explanation being that in this case the rubber sound was bent back in the drainage tube. In this case a pressure up to 400 mm. Hg. was recorded on the manometer without evoking any sensation, and it is unlikely that even a fibrous gall bladder could withstand this pressure without some distension of its walls.

The results are shown in Table I.

The following conclusions are suggested by this small series of observations.

The gall bladder is entirely lacking in temperature sensation, and in sensibility to light touch. In this it resembles the whole of the alimentary canal from the cardiac sphincter to the inner end of the anal sphincter, the œsophagus being sensitive to temperature but not to touch.<sup>1</sup>

Distension of the walls of the gall bladder produces a sensation varying from extreme discomfort to actual pain. Patients find difficulty in expressing in words the exact nature of the sensation, but say that it is very unpleasant indeed, and is more like that of indigestion than any other pain. Nausea was present in four of the six cases, but vomiting did not occur.

The pain produced by distension was localised roughly to the region of the gall bladder in four cases out of six, once to the xiphisternum, and once to the left hypochondrium. The localisation was in every case rather vague, and the site of the pain was indicated with the whole hand rather than with one finger.

Referred pain only occurred in one case, and in this it spread from the left hypochondrium, round the costal margin to the left scapula.

The observations in Case III. suggest that pain may be occasioned, not by the active contraction of the muscles of the wall of the gall bladder, but by the passive stretching of all its tissues produced by an internal tension which the muscle fibres are unable to overcome. This view was brought forward



some years ago by Hurst in connection with œsophageal and gastric pain,<sup>2</sup> and is in agreement with some experimental findings of Payne and Poulton in the case of the œsophagus.<sup>3</sup> The musculature of the gall bladder is but a feeble network of sparse fibres irregularly arranged, and it is unlikely that it could exert a pressure in any way comparable to that of the wall of the ureter or the intestine. Yet the pain of "gall-stone colic" is, if possible, more severe than that due to obstruction of these powerful tubes.

It appears probable that stones in the gall bladder, except when they cause obstruction or an acute infection, are unlikely to give rise to symptoms by which their presence can be detected apart from the dyspepsia with which they are associated; that pain due to obstruction of the cystic duct occurring without infection may be very vaguely localised to the upper abdomen, and is unlikely to be accompanied by referred pain; and that accurate localisation of pain in gall-bladder disease is an indication of involvement of the parietal peritoneum by an infective process.

#### THE PROBLEM OF INFECTION

The incidence of gall stones can only be satisfactorily explained on the view that there are two factors implicated, one biochemical, the other infective. It is unnecessary here to enter into the arguments for and against the purely biochemical view, but sufficient to say that the great bulk of evidence points to infection of the gall bladder as a precursor to stone formation in most cases. The organisms commonly involved are streptococci, and bacilli of the colon typhoid group.

Organisms may find their way into the cystic duct from the duodenum. C. J. Bond<sup>4</sup> has demonstrated that particles of indigo taken by the mouth may be recovered from the gall bladder in cases of cholecystostomy, thus showing that even inert particles may enter the bile duct from the duodenum in the reverse direction to the normal flow of its contents. But the epithelial lining of the gall bladder appears to be impervious to the passage of inert foreign particles of the size of bacteria. In three cats I injected a suspension of carmine directly into the lumen of the gall bladder, the cystic duct being tied. In the first two (experiments C. 3 and C. 8) no carmine could be detected by the naked eye in the gall bladder wall after death. In the third (experiment C. 12) sections were cut of the viscus, but no particles were found except in the lumen.

It is undeniable that cholecystitis may result from an

infection ascending from the duodenum by way of the bile ducts, as originally propounded by Naunyn, but it is doubtful whether the gall bladder is frequently infected by this route. In typhoid fever the bacilli are constantly found in the bile of the gall bladder, but these are being excreted from the liver, to which they have been carried by the portal stream. The infection of the gall bladder which occurs in this disease, and which is probably the cause of the stones which are an occasional sequela, is more likely to be due to organisms which have been carried to its walls by the general circulation, than to the penetration of its epithelium by those in the lumen. Rosenow introduced streptococci into the gall bladder, and was unable by this means to produce cholecystitis.<sup>5</sup> From without, infection may reach the gall bladder either by the blood stream or through lymphatic channels. Many observers have called attention to the lymphatic route. Sudler,<sup>6</sup> some twenty years ago, pointed out the intimate connection between the lymphatics of the gall bladder and those of the liver and pancreas. Graham<sup>7</sup> has more recently insisted on this lymphatic inter-relation, and has suggested that the gall bladder may in some cases be infected from the liver by this path. Braithwaite<sup>8</sup> has brought forward the possibility of infection reaching the gall bladder from the appendix by means of a retrograde flow along the lymphatic channels. The frequency with which a chronically inflamed appendix is found in cases of gall-bladder disease is a surgical *cliché*, but, as will be more fully discussed later, the discovery of two infective lesions should not necessarily lead to their association as cause and effect.

The spread of infection from one organ in the abdomen to the other by lymphatic paths has, except in the case of the pancreas, very little evidence of a practical nature to support it. The anatomy of the lymphatics has been investigated by Gerota's method, the injection of a coloured solution into the lymphatic plexuses of an organ or territory, by which means the efferent vessels from that territory are filled with the dye. By this method the existence of connections between the lymphatics of the gall bladder and those of the pancreas, stomach, duodenum, and appendix has certainly been proved. But while the injection of dyes is a useful anatomical method, it is of little value as a proof that infection may actually spread by these routes. It is difficult to estimate the pressure in the lymphatics, but, arguing by analogy from the smaller veins, and considering the diameter and structure of these lymphatic vessels, and the rate of flow of the lymph, it appears that it cannot amount to more than 1 or 2 mm. of mercury. In the investigations referred

to, the dye solutions have been injected at a much higher pressure, Braithwaite, for instance, using one of 6 to 8 mm. mercury and often more. The effect of such a method will be to fill the whole lymphatic system of the organ or tissue, and to force the solution along all the paths that are open to it, paths which would not come into play under normal or even pathological conditions. Where infection is conveyed in the body by lymphatic routes, the bacteria are carried by the wandering phagocytic cells, and not free in the lymph stream. The method used by Herring and Macnaughton,<sup>9</sup> in which insoluble particles of a neutral dye, comparable in size to bacteria, are injected, and their transference traced by microscopic sections, approximates far more closely to the conditions occurring in the body. These writers have shown that carmine particles are taken up by phagocytes in the same manner as tubercle bacilli, and carried by them to the same lymphatic glands. The method has also been used by Durham<sup>10</sup> and Bolton<sup>11</sup> to trace the absorption from the peritoneum.

During 1923 I used this method of injection of insoluble dyes in a series of experiments on cats, in order to study the interrelation between infective processes in the various parts of the alimentary tract. These investigations will be described at greater length elsewhere, but the general conclusions may be insisted on here. Particles of an insoluble dye are carried from the site of injection to the nearest group of lymphatic glands, and there fixed by the phagocytic cells lining the lymph sinuses. Where the dye is injected directly into the terminal lymph glands of the mesentery, or where it arrives in greater quantities than the glands can deal with, the particles are carried along the thoracic duct to the blood stream, and distributed by the general circulation. I obtained no evidence of the transference of particles from the territory of one organ to another, or of their carriage in the lymph stream in a retrograde direction. Once the phagocytic cells have taken up their load of particles, they move in a centripetal direction only. It appears then that the infection of one part of the alimentary tract by direct lymphatic spread from another part is improbable. The pathological evidence in support of such a hypothesis is no better than the experimental. The lymphatics of the different abdominal organs drain into a subperitoneal plexus, whence collecting vessels carry the lymph to the regional glands. Were infection by a retrograde flow of common occurrence, we should expect to find its earliest and most marked effects on the peritoneal coat of the organ. This condition is never seen in the gall bladder, stomach, or duodenum, and probably does not occur. From

this generalisation the pancreas must be excepted. It is interpolated in the path of the lymphatic channels from the liver, gall bladder, stomach and duodenum, and in all animals except orthogrades it lies between the layers of the mesentery. Invasion of the pancreas by the lymphatic route has been established by Deaver<sup>12</sup> as a common occurrence secondary to infection of the upper alimentary organs, and especially the gall bladder. In the experiments quoted above I obtained evidence of the permeation of carmine-laden cells from the mesenteric glands along the lymphatic channels between the pancreatic lobules.

We must look to the blood stream as the route by which infection generally reaches the gall bladder. Rosenow,<sup>13</sup> in a large series of animal experiments, has shown that streptococci isolated from the teeth, tonsils, appendix, gall bladder, and from gastric ulcers, in man, were able to produce cholecystitis in animals when injected intravenously. He found that those organisms which were grown from human gall bladders produced the highest proportion (80 per cent.) of gall-bladder infection in animals, a characteristic which he calls "elective localisation."

It seems unnecessary to postulate a strain of the streptococcus with a special affinity for the gall bladder, since it is more probable that organisms, whatever their source, which have established themselves in an organ, will develop a special immunity to the antibodies produced by the cells of that organ, and will, when injected into animals, find it more easy to establish themselves in the same organ than in a fresh tissue. These organisms may, in the case of the gall bladder, have come in the first instance from the teeth, tonsils, or nasal sinuses. But there is no evidence that infected teeth or tonsils are commoner in gall-bladder patients than in the general population. The conditions that appear to accompany gall stones with surprising regularity are flatulent dyspepsia and constipation.

When these patients are investigated by fractional gastric analysis, they show an unusually high incidence of achylia or marked hypo-acidity. Bonar<sup>14</sup> found that 49 per cent. of patients with gall stones had achlorhydria. Rydgaarde<sup>15</sup> found achylia in 47 per cent.—a strikingly similar figure, and a much higher proportion than is found in any condition except Addison's anæmia and gastric carcinoma.

It is often assumed that the gastric disturbance and achylia are secondary to the gall stones, but it is at least equally probable that in this condition we may have the explanation of an infection to which the cholecystitis, and, *post hoc*, the gall stones may be traced.

In 1922 I investigated the duodenal contents of six apparently normal students, who were at that time dressers in the surgical wards, and found that, while the bile in each case was sterile, streptococci were found in the duodenum in four out of the six.

The results are shown in Table II.

TABLE II.  
SIX NORMAL STUDENTS.

Initials.	Resting Gastric juice.		Duodenal contents.		Bile.
	Free HCl.	Total acid.	Microscope.	Culture.	Culture.
R. B. F.	26	40	Many leucocytes.	Sterile.	Sterile.
J. A. C.	22	32	Few leucocytes.	<i>Streptococcus longus.</i>	Sterile.
G. T. H.	42	47	0	<i>Streptococcus longus.</i>	Sterile.
H. C. C. T.	Had drunk water. 0	16	0	<i>Strep. long.</i> <i>Staph. albus.</i>	
L. H.	0	7	Many leucocytes.	Hæmolytic <i>Strep.</i>	Sterile.
A. T.	Not done.		0	<i>Enterococcus.</i> <i>Strep. long.</i> <i>B. proteus.</i> <i>Staph. aureus.</i>	Sterile.

These organisms were not numerous, as is the case in (Addison's pernicious) anæmia, but were probably some that had entered with the food and were passing through, rather than established in, the duodenum.

Streptococci are present in most uncooked foods. Biddle tells me that, in routine examinations at the Clinical Research Association, they are commonly found in milk, ice-cream, flour, shell-fish, vegetables, and fruit. Frost and Bachmann<sup>16</sup> state that a hæmolytic streptococcus can be isolated from 28 per cent. of cultures of high-grade milk. These streptococci are continually being swallowed by everyone, and where there is an absence of the "gastric germicidal barrier" in achylia, or where the food is only exposed for a short time to the action of the gastric juice, they will pass on unharmed into the intestine.

Campbell, in investigations on normal students, found that under the rather unappetising conditions of the test-meal the concentration of hydrochloric acid did not reach the germicidal level established by Knott<sup>17</sup> for streptococci in sixteen out of sixty-two, more than 25 per cent.).<sup>18</sup> In about half the



patients with gall stones the gastric barrier does not exist, while in many others it is inefficient. In achlorhydria the pylorus is permanently relaxed,<sup>19</sup> allowing food to pass rapidly into the duodenum. But even with a normal gastric secretion, liquid foods pass rapidly through the stomach. Water taken by mouth commences to enter the duodenum in thirty seconds.<sup>18</sup> Cushing found that a glass of milk could be recovered from a jejunal fistula a few minutes after swallowing, with its bacterial flora remaining practically unchanged.<sup>20</sup> In February 1924 I swallowed half a pint of milk containing bacillus coli, and long and short streptococci, having previously passed a tube down to the third part of the duodenum, and verified its position by the x-rays. Both organisms, absent from the specimen of duodenal contents taken before the milk was swallowed, were recovered by Dr. Biddle from consecutive duodenal specimens withdrawn within five minutes of swallowing the milk.

It appears certain that streptococci enter the intestines of all individuals from time to time. The chief difference in patients with gall stones is that in at least half of them this is a daily occurrence.

These pathogenic organisms which have passed the stomach are not destroyed in the intestines, for milk-borne streptococci can be identified in the fæces.<sup>21</sup> But the most important point to determine is whether they merely pass through the alimentary canal, or whether they can penetrate its walls and enter the circulation.

It has long been recognised that the flesh of animals killed while fasting is less liable to decompose than that of animals killed after a meal. Laboratory horses used in the preparation of remedial sera are starved before bleeding, to ensure that the serum shall be sterile. Ford<sup>22</sup> found that various bacteria could be obtained in 60 per cent. of cultures from the liver and kidneys in healthy dogs, cats, rabbits, and guinea-pigs. The fact that alimentary bacteria gain entry into the general blood stream during the digestion of a meal has also received silent tribute from surgeons in the accepted ritual of pre-operative starvation.

More direct evidence is not lacking. Tubercle bacilli are constantly found in the mesenteric glands of children in whom close inspection fails to reveal any evidence of a recent or healed lesion anywhere in the alimentary tract. Ruffer,<sup>23</sup> and later Nicholls,<sup>24</sup> have demonstrated the constant presence of bacilli in the mesenteric glands of healthy rabbits. Desoubry and Porcher<sup>25</sup> showed that during the digestion of fats many bacteria of all sorts are found in the blood and the chyle.

Nicholas and Dercas<sup>26</sup> fed dogs on fatty soup mixed with tubercle bacilli, and killed them three hours later. The chyle from the thoracic duct was injected into guinea-pigs, and tubercle bacilli were recovered. These experiments have been repeated in many different animals by Ravenel,<sup>27</sup> V. Behring and Roemer, Bisanti and Panisset, Ficker, Oberwarth and Rabinowitch,<sup>28</sup> and bacilli have been obtained in many instances from the blood as well as the chyle. Moody and Irons,<sup>29</sup> feeding dogs on *B. pyocyaneus* and *B. prodigiosus*, were unable to recover the organisms from the tissues; when, however, these two bacilli were injected directly into the duodenum, they were recovered from the thoracic duct in certain cases after one or two hours. Calmette,<sup>30</sup> by killing guinea-pigs, goats, and cattle, at different periods after an infected meal, has found bacilli in the central lacteal of the villi, lying in the substance of leucocytes.

From the physiological aspect it is generally denied that particulate matter can pass through the walls of the healthy alimentary canal. Some experiments of Bradley and Gasser<sup>31</sup> suggest that this may not be true in the case of fats. These observers fed dogs through a tube with an emulsified mixture of olive oil and petroleum, and recovered the same oils in the same proportions from the thoracic duct. I investigated the same question by feeding carmine, intimately mixed with their food, to mice and cats. Twelve mice were used, and killed at intervals up to a year after commencement of carmine feeding. Five cats were killed after one month of the dye. In some of the animals, finely powdered glass was added to the food as well as the carmine. Numerous sections of all parts of the alimentary canal and the mesenteric lymph glands, as well as of the liver, spleen, pancreas, and gall bladder, were carefully searched under the high power, but no evidence could be found that carmine had been taken up by the alimentary canal, or had entered the tissues. Though I was unaware of the fact when I undertook these investigations, Lister made similar observations on the mouse in 1858,<sup>32</sup> and experiments in the feeding of dyes have been carried out by several observers since, with uniformly negative results.

I next examined the effect of stasis on the absorption of carmine from the alimentary tract. For this purpose I excluded various parts of the alimentary canal in cats by an anastomosis above and below, the excluded portion being left in connection with its blood supply and lymphatic drainage. The excluded loop was ligatured at both ends, and a suspension of carmine in saline solution was put into it, a condition of artificial stasis

being produced, which lasted as long as the animal was kept alive, the usual period being forty-eight hours. Observations were made by these means in the case of the stomach, duodenum, small and large intestines, appendix, and gall bladder. In the stomach, a few carmine-laden cells were found in lymphatic nodules lying under the mucous coat near the pylorus. In the case of the appendix, similar cells were found in the regional glands, and in the case of the colon, lying in the mesenteric



FIG. 3.

Small intestine of frog (oil immersion). Carmine particles, shown in black, are seen lying free in the lumen of the gut, and also in the substance of large cells situated at the base of the epithelial layer.

lymphatic channels, showing that carmine had been taken up from the lumen of these organs as a result of obstruction to the passage of their contents. In the gall bladder and small intestine the results were negative.

In six decerebrated frogs the middle portion of the intestine was isolated by two ligatures, and the closed portion filled with carmine suspension. The frogs were killed at the end of twenty-four hours. In one of these frogs very striking evidence of the passage of carmine through the intact epithelium of the alimentary canal was obtained, particles of the dye being found

in the substance of large cells lying at the base of the epithelial layer, and abutting on the lymph channels (Fig. 3). It appears then that, while under normal conditions inert particles do not pass through the walls of the alimentary canal, at any rate in the higher vertebrates, they may do so in conditions of stasis.\*

I also made four experiments upon cats with living streptococci. In two cases an emulsion of hæmolytic streptococci was injected into the upper part of the intestine. One animal was killed after five hours; upon opening the abdomen, the ileocæcal lymphatic glands were found to show bright patches of subperitoneal hæmorrhage, though the intestine itself was of normal appearance. Cultures from the glands and thoracic duct proved sterile. The second cat was killed after  $3\frac{1}{2}$  hours. In this animal streptococci were grown in cultures from the ileal, colonic, and ileocæcal groups of glands, but only from the latter in considerable quantities. These were not proved to be hæmolytic.

In two other cats a long ileocæcal loop was excluded, and the streptococcal emulsion injected into the closed loop. These animals were killed after five hours. The occluded loop of gut was of normal appearance, not distended, and with smooth, glistening peritoneal coat. When opened it showed no visible lesions, nor were any seen in microscopic section. In both, the nearest group of lymphatic glands again showed patches of subperitoneal hæmorrhage. In one of these cats all cultures were sterile, in the other hæmolytic streptococci in pure culture were grown from the thoracic duct and the mesenteric lymph channels.

\* Keith,<sup>33</sup> discussing in 1915 the microscopical changes found in the large intestines of patients suffering from intestinal stasis, pointed out that in many of these specimens, large cells containing brown pigment are found in the submucous coat of the colon. Very similar cells are found lining the blood spaces in the liver of the frog, and Pickof<sup>34</sup> has shown that the granules of pigment in these cells contain no iron. In the course of examining the livers of the six frogs used in my experiments, I was struck by the difference in the amount of pigment in these cells, some being absolutely black, others containing a few granules only. In the cells containing a few pigment particles, grains of carmine were also found. It occurred to me that the probable explanation of these appearances is that the intestinal epithelium of the frog, even under normal conditions, allows occasional particles of foreign matter to pass through with the food, and that these are then carried to the liver by the scavenging leucocytes, and there fixed by the endothelial cells. When some of the cells are packed with pigment, other fresh ones take up the task, and it was these cells which were at the time actively dealing with foreign particles which fixed the carmine granules which had been taken up from the alimentary canal. According to this view, the alimentary epithelium of the frog is intermediate between that of simple animals like Hydra, which ingests all foreign particles, and that of the mammals, which allows none to pass. But where, in man, stasis has damaged this acquired function of exclusion, particles do pass through, and are fixed by cells in the submucous coat. I discussed this possible explanation of the pigment granules found in stasis in human beings with Sir Arthur Keith, and he agreed that the explanation was quite a possible one.

These experiments suggest that streptococci had passed through the intestinal walls and had entered the lymphatic channels, where they had produced destructive changes in the blood vessels, causing subperitoneal hæmorrhage, but had themselves been destroyed. In one case the organisms had survived all the lymphatic barriers, and had reached the thoracic duct in a living condition.

It appears probable, therefore, that a condition of stasis will increase the entry of bacteria from the alimentary canal into the general circulation. Another factor which will also have this effect is a diet deficient in accessory food factors, as has been shown by McCarrison in several series of animal experiments.<sup>35</sup> Bacteria enter the circulation during digestion even in normal individuals, but their number is so small that they are rapidly exterminated by the defence mechanisms of the body. Where pathogenic bacteria have free access to the alimentary canal owing to a breach in the gastric defence, and where their absorption is also favoured by stasis, or a diet qualitatively deficient, they will arrive in the tissues in varying numbers, with a capacity for mischief depending on their virulence and the resistance, general or local, of their host. These two conditions are present in gall-stone patients, and it appears unnecessary to demand a local lesion, where it is probable that the whole alimentary canal is the infecting agent.

Teeth and tonsils are undoubtedly the source of the bacteria in some cases of gall-bladder infection, and will require treatment. The appendix is very frequently found to show evidence of present or recent infection, and such an appendix is rightly removed. Organisms from the appendix may have caused the cholecystitis; if so, they have been carried by the blood stream and not by direct lymphatic spread. But the appendix may be, in such cases, not the main source of the infection which has damaged the gall bladder, but additional evidence of a general intestinal infection. The appendix is the sentry of the ileocæcal angle, as the tonsil is that of the fauces. Whenever a fresh pathogenic organism appears in either situation, the sentry suffers. In the first few days of typhoid fever, a tender appendix gives evidence of the struggle of the lymphatic system against a general invasion of the body by the typhoid bacilli from the alimentary canal. In gall stones, an inflamed appendix should at any rate suggest that there is a general failure of the motor and secretory functions of the alimentary tract, which will require treatment after the main lesion has been dealt with by surgery.

## THE CHOICE OF OPERATION IN GALL STONES

Carrying the investigation of the absorption of particles from the alimentary canal one step further, we find that if carmine be injected into the mesenteric lymph glands in a greater quantity than they can deal with, the granules are carried into the blood stream. Only a small proportion of the carmine injected will escape fixation by the lymphatic glands, and the situations in which it may alone be possible to demonstrate the presence of the pigment are the endothelial cells of the spleen and the liver. In three experiments upon cats (C. 11, NS. 6, and NS. 10), 1 c.c. of a suspension of carmine was injected into the lacteals in the mesentery of the ileum and into the glands at the base of the mesentery. In each of these experiments the presence of cells containing carmine in the spleen showed that some of the pigment had reached the general circulation through the thoracic duct. In experiments NS. 6 and 10 this amount had been small, and no carmine could be demonstrated in the gall bladder. In experiment C. 11, carminiferous cells were found in sections of the gall-bladder wall. In three consecutive sections, 5 such cells were found in the mucous coat, 9 in the musculo-fibrous coat, and 3 in the subperitoneal coat. In another experiment (NS. 3) a suspension of 0.5 gm. carmine in 20 c.c. saline was run very slowly into a radicle of the portal vein in the mesentery of the ileum. Such an amount was greater than could be fixed by the endothelial cells of the liver, and carmine again passed into the general circulation, as shown by its presence in the spleen. In 45 consecutive fields under the high power, carmine was found in the following situations in the gall-bladder wall:—mucous membrane 4, musculo-fibrous coat 3, subperitoneal coat 11 (Fig. 4). The stomach in experiment NS. 3 showed 8 carmine cells in the mucous membrane, and 3 in the muscular coat in 100 consecutive fields. In the stomach in experiment C. 11, 8 such cells were found in the mucous membrane and none in the wall.

While too great weight must not be attached to these findings, it appears that particles borne by the blood stream tend to lodge in the outer coats of the gall bladder more often than in the mucous membrane. In the alimentary canal the reverse is seen. It is in these respective situations that the earliest signs of inflammatory processes are found; in the stomach, small ulcers involving the mucous coat only, in the gall bladder a mural infection with an apparently healthy mucous membrane.

A condition of infection in the coats of the gall bladder,

leading to a fibrosis, appears to be the precursor of gall stones in the majority of cases, even if we admit the possibility of the occasional formation of stones in a healthy gall bladder owing to a disorder of cholesterol metabolism. In 1920, both MacCarty in America<sup>36</sup> under the name of "strawberry gall bladder,"\* and Gosset, Loevy, and Magron in France,<sup>37</sup> described a condition in the gall bladder in which multiple collections of a cholesterol ester, visible to the naked eye, are found in the submucous



FIG. 4.

Part of the gall bladder of the cat in experiment C. 11. Carmine had been injected into the mesenteric lymph glands 2/8/23. The cat was killed 4/8/23. Carmine granules (black) are seen in the fibrous coat of the gall bladder.

coats. It has been pointed out that these submucous collections tend to become pedunculated, lose their attachment to the wall, and form the nucleus of a gall stone. Chauffard has cited the work of the French observers in support of the biochemical theory of gall-stone formation. A very careful study of this condition was made in 1922 by Boyd,<sup>39</sup> who found evidence of infection in every one of fifty-two human gall bladders in which deposits of cholesterol were demonstrable in the wall. "When

\* Moynihan described this condition clinically in 1909.<sup>38</sup>

we speak of infection as a factor in the production of gall stones," he writes, "we should think of the bladder wall rather than of the free bile."

Drainage of the gall bladder in the treatment of gall-stone disease is advocated on three main grounds. That the gall bladder plays a necessary and important part in the physiology of digestion; that, if free drainage is provided, any infection present will subside, and leave a healthy and useful gall bladder; and that, after removal of the stones and drainage of the gall bladder, any recurrence of trouble is unlikely.

The traditional view of the function of the gall bladder in digestion is that it serves as a reservoir in which is stored the bile secreted by the liver during the period between successive meals.<sup>40 41</sup> When the acid chyme passes into the duodenum the gall bladder contracts, forcing out its store of accumulated bile. The mechanism by which this contraction is brought about is variously stated as being a nervous reflex, or the action of secretin. Melzer<sup>42</sup> stated that a solution of magnesium sulphate applied to the duodenal papilla causes contraction of the gall bladder, and Lyon<sup>43</sup> believed that by introducing such a solution through a duodenal tube it was possible to obtain specimens of gall-bladder bile in human subjects.

The recent experimental work of Auster and Crohn,<sup>44</sup> of Rous and Macmaster,<sup>45</sup> and of Bassler, Luckett, and Lutz,<sup>46</sup> as well as the reports of clinical workers studying the duodenal bile by Lyon's method, have largely modified these views. Animal experiments have established the following facts. The gall bladder is not necessarily distended in a fasting animal, or empty in a digesting one. It does not contract during digestion. In dogs its contents are often not expelled into the duodenum over a period of seventy-two hours. The flow of bile into the duodenum is not inhibited by fasting. Magnesium sulphate, peptone, and dilute hydrochloric acid in the duodenum, all cause an increased flow of bile from the duodenal papilla, but do not produce emptying of the gall bladder. Clinical experience with the duodenal tube has shown that the three types of bile described by Lyon are not recovered as the result of irrigation with magnesium sulphate.<sup>19</sup> The main function of the gall bladder is that of a safety-valve, a distensible by-pass in a rigid system of tubes, whose capacity is greatly increased by its power of concentrating the bile eleven times, and whose purpose is to minimise extremes of pressure when the liver is secreting rapidly and the sphincter of Oddi is closed. It appears, therefore, that the gall bladder plays no essential part in digestion, and no



evidence has been brought forward that digestion is in any way impaired by its removal.

The morbid processes which may follow in the train of a persistent gall-bladder infection do not need enumeration, but the fact that pancreatitis is almost invariably the result of such an infection, while 27 per cent. of cases of cholecystitis show changes in the pancreas,<sup>47</sup> is in itself sufficient reason for the removal of a gall bladder that is suspect. The existence of stones makes it highly probable that there is an infection, active or slumbering, in the gall-bladder wall. Rosenow has shown that in cholecystitis the bile is often sterile, while streptococci may be grown from the wall. In view of this fact, the statistics of Rovsing,<sup>48</sup> that in a series of 530 cases of gall stones the bile was sterile in about 60 per cent., are no proof that the gall bladder was uninfected in these cases. Nor is it possible to say by naked-eye examination that infection is not present.

Drainage of a cavity may reasonably be expected to result in the eradication of an infection where the organisms are mainly in the lumen of the cavity, and where the outlet is obstructed. Where the infection is resident in the walls, even prolonged drainage may fail to effect a cure. An infection of the gall bladder resembles one of the appendix rather than of the urinary bladder.

The third reason which has been advanced in favour of drainage of the gall bladder, that recurrence of stones after such an operation is uncommon, will not bear close scrutiny. Individual surgeons rarely see the recurrences in their own cases. Tanner,<sup>49</sup> in seventy-six cases of cholecystostomy whose after history was investigated, found that a second operation for stones had been necessary in 9 per cent. When the frequency with which stones may exist without producing symptoms is taken into account, it will be realised that the percentage of recurrences may well have been higher.

It is suggested, therefore, that cholecystectomy should be the operation of choice in all cases of gall stones. Pathological considerations demand the removal of a gall bladder which is presumably infected, while the physiological value is insufficient to justify its retention under such circumstances.

I wish to express my thanks to Sir Alfred Fripp, Mr. Fagge, and Mr. Rowlands for allowing me access to their cases in the wards; to Dr. Hurst for advice and helpful criticism; and to the dressers who were the subjects of the duodenal experiments.

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# CIRCULATORY CHANGES IN WOUNDED SOLDIERS, WITH SPECIAL REFERENCE TO THE INFLUENCE OF DRUGS USED FOR THE PRODUCTION OF ANÆSTHESIA

By GEOFFREY MARSHALL, M.D., Assistant Physician to Guy's Hospital.

It is now more than seven years since the observations were made on which this paper is based. The War provided a mass of clinical material from which important lessons were learned, and one of the most striking of these was the effect of various forms of anæsthesia on the mortality following surgical operations. There are still those who do not realise that the vast bulk of the deaths due to anæsthetics do not take place on the operating table, but hours after the patient has been returned to bed.

These studies were undertaken with a view to devising measures which would reduce the mortality amongst men suffering from severe wounds, *i. e.* from the effects of hæmorrhage, shock or a combination of the two. The clinical features of a case suffering from loss of blood are well known. In "shock," as I pointed out in 1917,<sup>7</sup> it is common to find in addition to the pallor, rapid pulse and reduced blood-pressure, an unclouded mentality, a lowered surface temperature and concentration of the blood in the capillaries of the extremities. It was necessary to subject these men to early operation if they were to be preserved from a fatal extension of sepsis, but early operation was a dangerous procedure and in itself caused a heavy death-rate. These men, whether they were suffering from shock or hæmorrhage, were particularly susceptible to what has been termed "operation shock," and my first endeavour was to show how this might be mitigated.

## HISTORICAL

In civil practice it was known that in certain operations attended by a heavy mortality the nature of the anæsthetic was one of the most important factors; thus there had been very few recoveries after resection of gut in children suffering from intus-susception until Fairbank and others reported the advantages of spinal anæsthesia for this operation. The success of this technique was attributed by Gray to the effect of the regional anæsthesia in preventing afferent impulses from reach-

ing and exhausting the upper nervous centres in accordance with Crile's theory<sup>3</sup> as to the production of shock. In our view the success is due to the avoidance of the volatile anæsthetics of the ether chloroform series.

#### NATURE OF OBSERVATIONS AND TECHNIQUE

Records were made of the pulse-rate, blood-pressure (diastolic and systolic) and the percentage of hæmoglobin in the capillary blood of wounded men before operation; blood-pressure and pulse-rate were recorded every two and a half minutes during the course of the operation, and at intervals of about half an hour for two or three hours after its conclusion. The hæmoglobin estimations were made at longer intervals. Blood-pressure was recorded by means of a Riva-Rocci sphygmomanometer with auscultation over the brachial artery. Hæmoglobin concentration was determined by the Gowers-Haldane hæmoglobinometer, the necessary carbon monoxide being obtained by heating a mixture of sulphuric and oxalic acids. Control observations showed that the variable error did not exceed four per cent.

#### SPINAL ANÆSTHESIA

From considerations referred to above it was generally thought that spinal anæsthesia would be the method of choice for operations on men wounded in the lower extremities. In fact this proved a safe procedure when I was working at a base hospital. In the clearing station an unexpected phenomenon was met with, a large proportion of the patients suffered a great fall of blood-pressure and presented an alarming picture of collapse within fifteen minutes of the injection. The signs were those of cerebral anæmia, pallor, sweating, nausea, retching, vomiting and loss of consciousness, occasionally extreme restlessness and in one case convulsions. I saw two cases in which the collapse terminated fatally.

My previous experience at the Base led me to suspect that the shorter interval of time elapsing between the infliction of the wound and the administration of the anæsthetic was an important factor in the occurrence of syncope. Analysis of cases showed this to be correct; in no case was there syncope or a serious fall of blood-pressure when the interval between wounding and the intrathecal injection of stovaine exceeded forty hours. Amongst the cases in which this interval was shorter, rather more than half showed a considerable fall of blood-pressure with some of the symptoms referred to above shortly after injection of the drug. The problem was now to

discover why there should be a big fall of blood-pressure in some of these short-interval cases and not in others. As the investigation proceeded a remarkable fact came to light: the serious falls of pressure were produced only in those patients whose blood was dilute before operation. The percentage of hæmoglobin in the blood of healthy unwounded soldiers ranged from 97 to 120 per cent., with an average of about 110 per cent.,

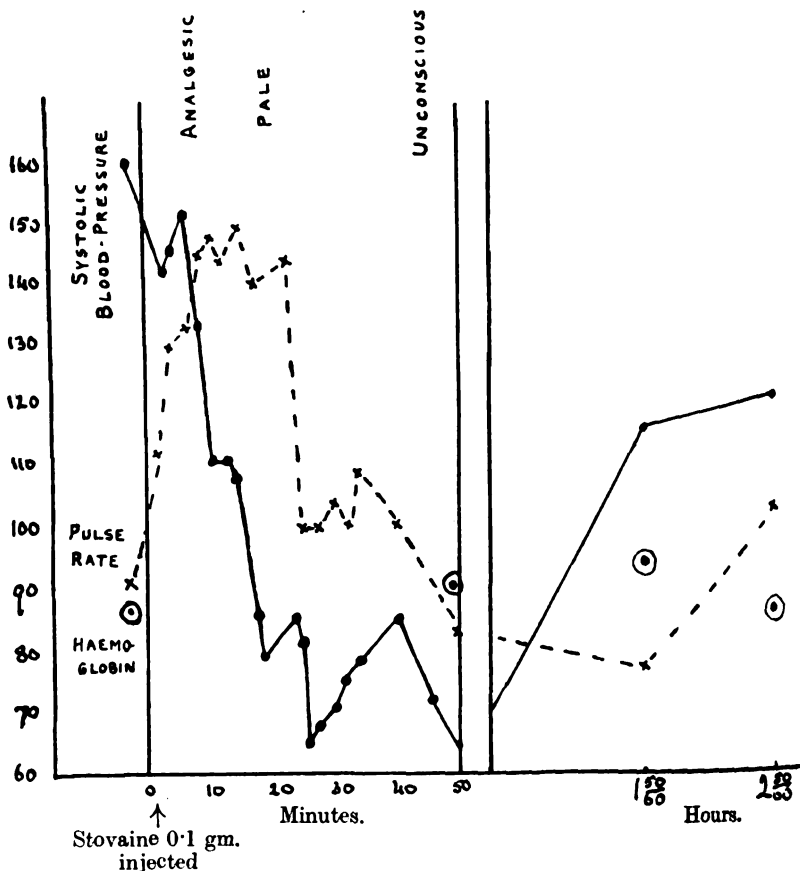


CHART I. SPINAL ANÆSTHESIA. Case No. 2. Shell Wound Leg, 5 hours. Operation: Ligation of Popliteal Artery. Hæmoglobin 86%.

when compared with the sample which I used for all my determinations. On examining the records of my cases I found that if I divided them arbitrarily into two groups, those with a hæmoglobin percentage of 100 or over, and those with a percentage of less than 100, the serious falls of blood-pressure occurred only in the latter group.

These points are illustrated by an analysis of fifty consecutive cases of soldiers wounded in the lower extremities operated on at a clearing station under spinal anæsthesia.

Case.	Interval.	Hæmo- globin.	Initial B.P.	Symptoms.	Fall of B.P.
1	23 hrs.	80%	135 mm.	Loss of consciousness.	60 mm.
2	5 hrs.	86%	160 mm.	Loss of consciousness.	96 mm.
3	36 hrs.	74%	136 mm.	Stupor.	64 mm.
4	36 hrs.	113%	150 mm.	Nil.	25 mm.
5	11 hrs.	95%	103 mm.	Pallor, retching.	49 mm.
6	14 hrs.	98%	115 mm.	Pallor, stupor.	59 mm.
7	16 hrs.	106%	115 mm.	Pallor.	22 mm.
8	25 hrs.	104%	117 mm.	Nil.	27 mm.
9	3 days.	102%	110 mm.	Nil.	7 mm.
10	26 hrs.	104%	115 mm.	Nil.	17 mm.
11	14 hrs.	100%	122 mm.	Nil.	9 mm.
12	3 days.	76%	131 mm.	Nil.	20 mm.
13	47 hrs.	81%	132 mm.	Nil.	28 mm.
14	13 hrs.	90%	128 mm.	Pallor, retching.	49 mm.
15	14 hrs.	110%	131 mm.	Nil.	33 mm.
16	7 hrs.	92%	134 mm.	Loss of consciousness.	86 mm.
17	20 hrs.	95%	122 mm.	Pallor.	52 mm.
18	23 days.	45%	105 mm.	Nil.	0 mm.
19	9 hrs.	94%	107 mm.	Pallor, stupor.	51 mm.
20	26 hrs.	91%	148 mm.	Nil.	38 mm.
21	24 hrs.	104%	145 mm.	Nil.	23 mm.
22	16 hrs.	102%	131 mm.	Nil.	6 mm.
23	14 hrs.	82%	90 mm.	Stupor, vomiting.	30 mm.
24	31 hrs.	76%	132 mm.	Stup., vom., convulsion.	76 mm.
25	20 hrs.	99%	124 mm.	Pallor, nausea.	44 mm.
26	17 hrs.	78%	144 mm.	Pallor.	23 mm.
27	30 hrs.	120%	126 mm.	Pallor.	31 mm.
28	36 hrs.	90%	134 mm.	Yawning, stupor.	77 mm.
29	9 hrs.	98%	57 mm.	Pallor, vomiting.	57 mm.
30	36 hrs.	83%	132 mm.	Pallor, vomiting.	68 mm.
31	15 hrs.	100%	158 mm.	Nil.	46 mm.
32	10 hrs.	100%	138 mm.	Nil.	15 mm.
33	7 hrs.	102%	131 mm.	Nil.	23 mm.
34	18 hrs.	92%	131 mm.	Pallor.	47 mm.
35	20 hrs.	98%	143 mm.	Nil.	35 mm.
36	44 hrs.	114%	140 mm.	Nil.	28 mm.
37*	14 hrs.	88%	132 mm.	Pallor.	31 mm.
38	48 hrs.	103%	143 mm.	Pallor slight.	35 mm.
39	21 hrs.	85%	123 mm.	Vomiting, loss of consc.	81 mm.
40	23 hrs.	101%	119 mm.	Nil.	1 mm.
41	15 hrs.	90%	125 mm.	Pallor, respiratory distress.	47 mm.
42	21 hrs.	110%	140 mm.	Nil.	15 mm.
43	12 hrs.	102%	138 mm.	Nil.	0 mm.
44	27 hrs.	102%	129 mm.	Nil.	10 mm.
45	3 hrs.	98%	100 mm.	Pallor, vomiting.	50 mm.
46	7 hrs.	90%	108 mm.	Vomiting, loss of consc.	65 mm.
47	10 hrs.	97%	182 mm.	Pallor, retching.	99 mm.
48	11 hrs.	98%	142 mm.	Retching, loss of consc.	74 mm.
49	21 hrs.	96%	137 mm.	Nil.	51 mm.
50	20 hrs.	102%	124 mm.	Nausea.	17 mm.

\* This patient became stuporous and pulseless when propped up on his return to bed.

In the above list the first column shows the serial number of the case, the second the interval between wounding and injection of stovaine, the third the percentage of hæmoglobin in the capillary blood before injection, the fourth the systolic blood-pressure before injection, the fifth the symptoms after injection, and the last column on the right shows the fall of systolic blood-pressure in mm. of mercury after injection of stovaine.

Let us group the above cases according to (1) the time interval between wounding and intrathecal injection, and (2) whether hæmoglobin concentration is above or below 100 per cent.

AVERAGE FALL OF BLOOD-PRESSURE IN GROUP WITH HÆMOGLOBIN.

Interval.	Less than 100%.	100% or over.
1 to 10 hrs.	72 mm. (7 cases).	19 mm. (2 cases).
11 to 20 hrs.	45 mm. (13 cases).	14.6 mm. (6 cases).
21 to 30 hrs.	57.5 mm. (4 cases).	17.7 mm. (7 cases).
31 to 40 hrs.	71 mm. (4 cases).	25 mm. (1 case).
41 to 50 hrs.	28 mm. (1 case).	31.5 mm. (2 cases).
51 hrs. to 23 days.	10 mm. (2 cases).	7 mm. (1 case).

We see that the serious fall of blood-pressure is produced only in the man who is given stovaine intrathecally within forty hours of being wounded, and whose blood is dilute. Of the twenty-two cases in this group all but three had untoward symptoms after injection: in only three was the fall of blood-pressure less than 35 mm., the greatest was 99 mm. There were sixteen cases, who had been wounded less than forty hours but whose blood was not dilute; amongst these only three showed symptoms after injection, one complaining of nausea and the other two exhibiting pallor only. The average fall of blood-pressure was 17 mm., the greatest 33 mm. It is of interest to note that determination of the concentration of the blood was the only method which enabled us to foretell accurately whether spinal anæsthesia would produce a serious fall of blood-pressure in a man suffering from recent wounds. Many of those in the first group showed no other signs of exsanguination, their appearance was normal, their blood-pressure and pulse-rate did not differ from those of patients whose blood showed a hæmoglobin concentration above 100 per cent. :—

	Group with Hb. less than 100%.	Group with Hb. 100% and over.
Average initial blood-pressure.	130 mm.	129 mm.
Range of initial blood-pressure.	90 to 182 mm.	115 to 150 mm.
Average initial pulse-rate.	105	88
Range of initial pulse-rate.	70 to 140	64 to 140

The question arises as to why intrathecal injection of stovaine should cause a big fall of blood-pressure in a patient who has bled less than forty hours beforehand. That it is not due simply

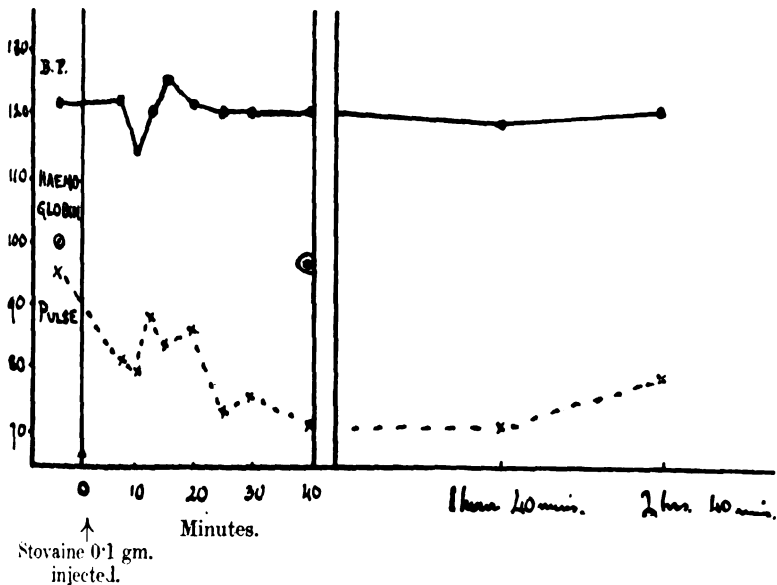


CHART II. SPINAL ANÆSTHESIA. Shell Wounds Thigh, 14 hours. Operation: Excision of Wounds. Hæmoglobin 100%.

to interference with intrathecal pressure is shown by the fact that in several cases where inactive samples of stovaine were injected and no anæsthesia resulted, the blood-pressure altered only a few millimetres of mercury. The most reasonable explanation would seem to be that the fall of blood-pressure results from the stovaine abolishing vasomotor control in the lower part of the body. This vasomotor paralysis produces little effect on the blood-pressure of a man whose blood is of normal composition; but in the man who has bled and whose blood-volume has been restored by rapid dilution with water from the tissues, the relaxed arterioles present so little resistance to a circulating fluid of reduced viscosity that the blood-pressure



may fall to a dangerous level. As a corollary to this hypothesis I would suggest that after an interval of about forty hours, although red cells and hæmoglobin are still deficient, the viscosity of the blood is restored by an increase in its colloid content.

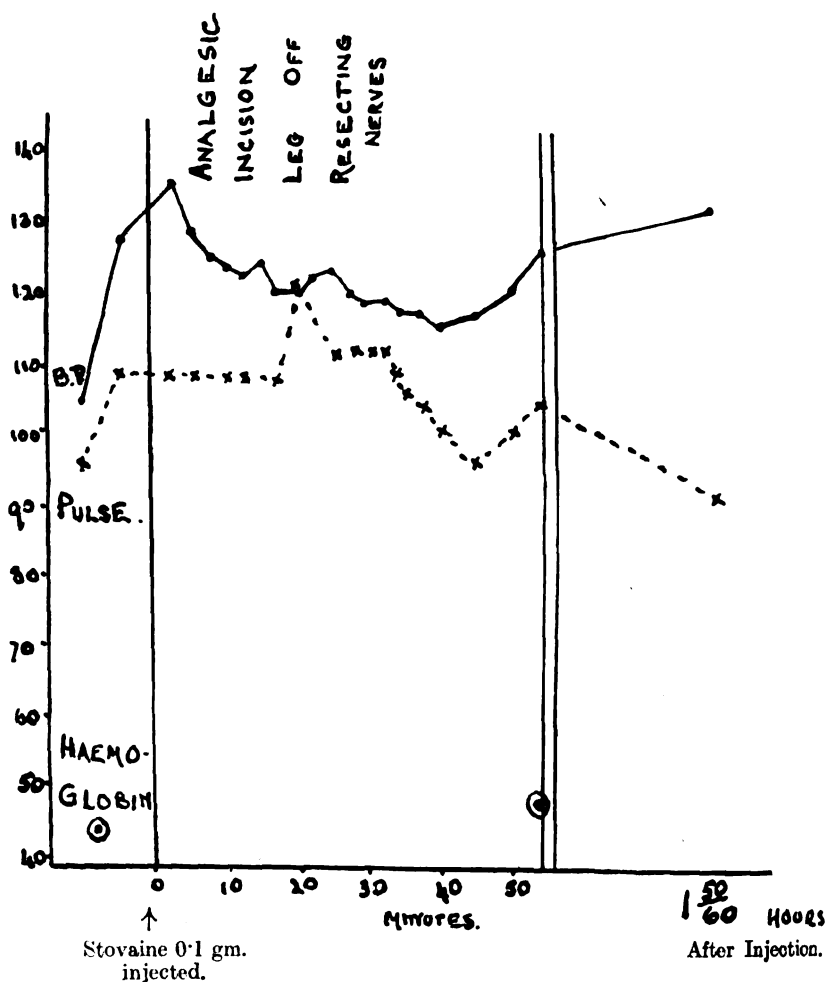


CHART III. SPINAL ANÆSTHESIA. Case No. 18. Shell Wound Thigh, 23 days. Operation: Amputation through Thigh.

I was unable to investigate this point owing to the difficulty of obtaining an efficient instrument for measuring viscosity.

#### EFFECTS OF ETHER AND CHLOROFORM

The conditions which lead to a fall of blood-pressure when ether or chloroform are administered are altogether different

from those in spinal anæsthesia. In the latter we see that blood concentration is the most important factor. The type of operation performed is of little significance, even amputation through the thigh producing no fall of blood-pressure either during or after the period of anæsthesia unless the patient be a short-interval case with dilute blood (see Chart III). Even in the latter case the blood-pressure will return to a normal level

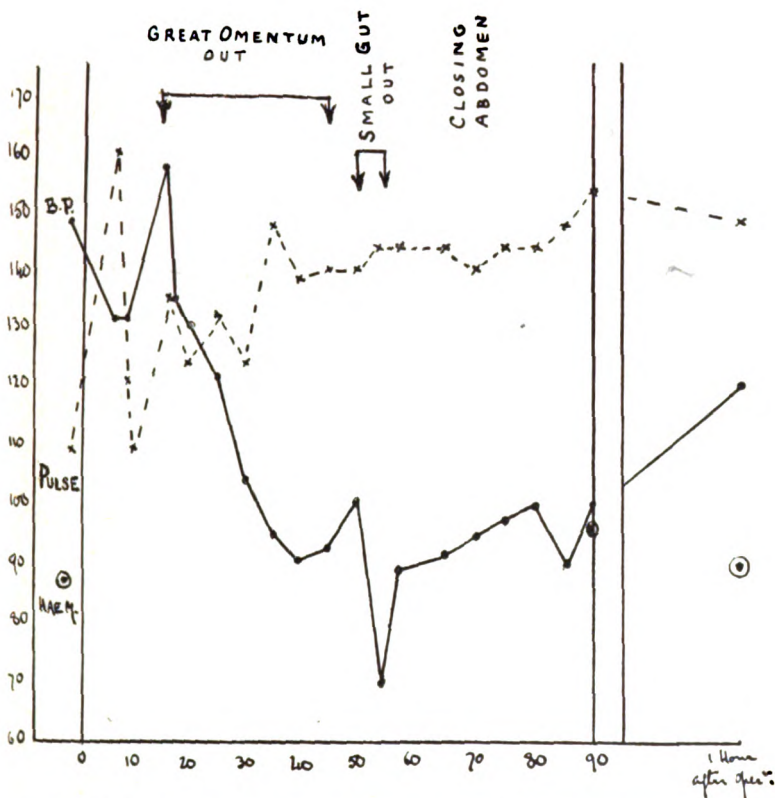


CHART IV. ETHER VAPOUR ANÆSTHESIA. G.S.W., Abdomen and Chest. Exposure of Gut outside Abdominal Cavity.

as soon after an amputation as after a much more trivial operation.

In the case of ether and chloroform blood concentration appears to be a factor of little importance when considering the effect these drugs will produce on the blood-pressure.

*Example.*—Private R. 21 years. Shell wounds chest wall and scrotum. 14 hours. Hæmoglobin 86 per cent. Blood-pressure systolic 87 mm. Pulse-rate 96. Operation under ether vapour anæsthesia, wounds excised and damaged testicle

removed. The blood-pressure actually rose during operation and remained above the initial level during the three hours following.

This case may be used to illustrate another point characteristic of the effects of ether and chloroform; *i. e.* that these drugs do not produce any serious fall of blood-pressure when carefully administered unless the patient be submitted to some other

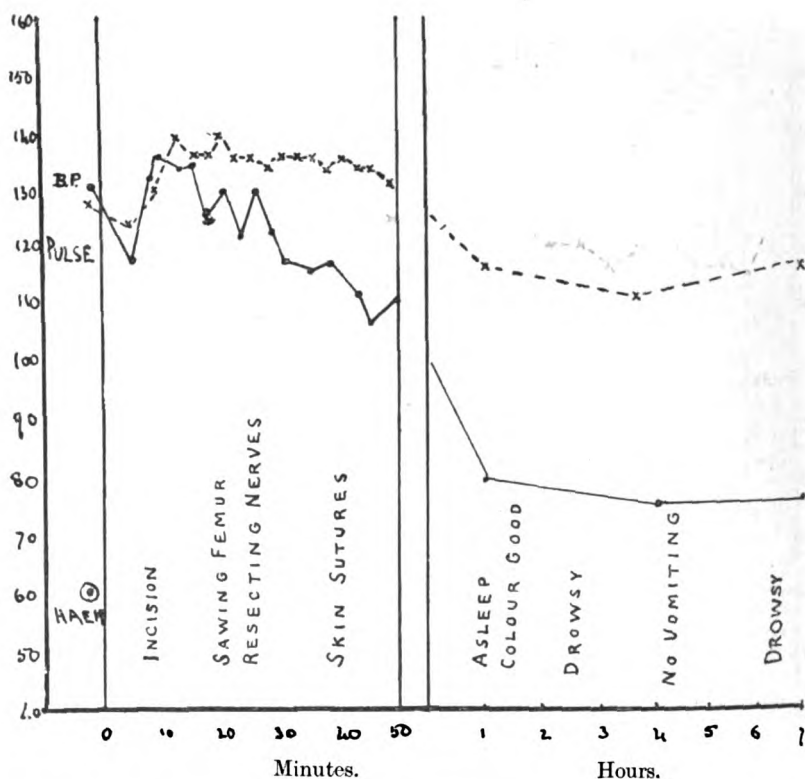


CHART V. CHLOROFORM VAPOUR ANÆSTHESIA. S.W. Leg, 8 days. 25/1/17.  
Amputation through Thigh. Death 10 hours after Operation.

interference, such as considerable trauma, while under their influence. If, instead of a comparatively trivial operation, this patient had been subjected to amputation of a limb, there would certainly have been a big fall of blood-pressure afterwards.

The only constantly reliable method of producing "operation shock" in one of the lower animals under ether or chloroform anæsthesia is to open the abdomen and expose considerable lengths of intestine outside the abdominal cavity. This pro-

cedure has the same effect when applied to man (Chart IV). When more than two or three feet of gut are so exposed, I found that the blood-pressure began to fall after a few minutes and continued to do so until the viscera were returned to the abdominal cavity. On the other hand, experience during the war showed that prolapse even of several coils of intestine through a wound

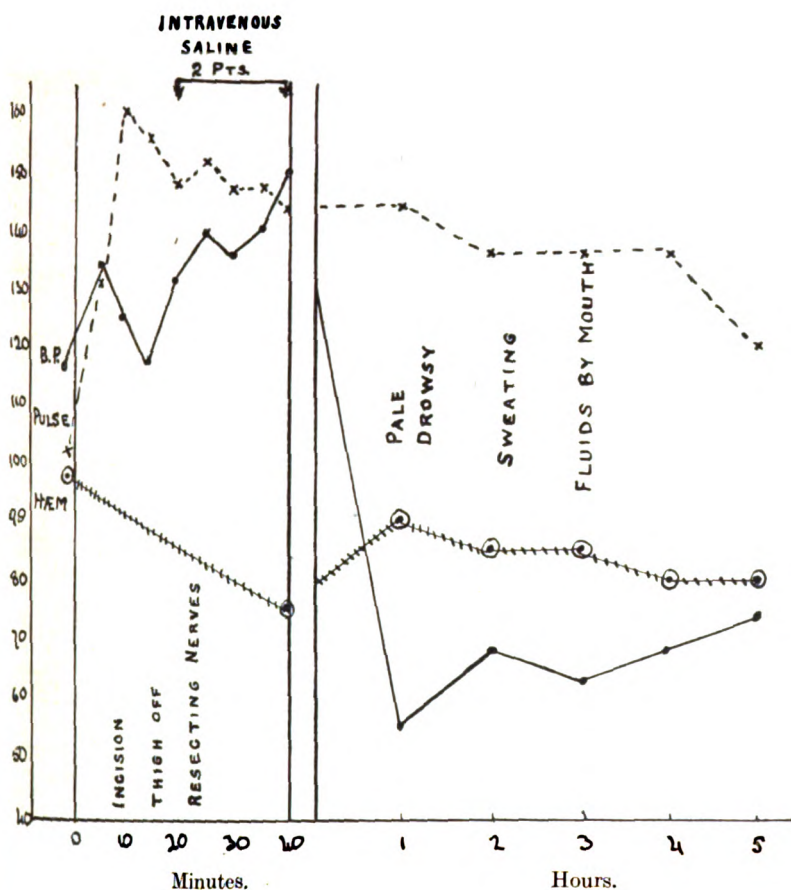


CHART VI. ETHER VAPOUR ANÆSTHESIA. Amputation through Thigh. 30/7/16D.

in the abdominal wall produced no such serious effect on the condition of a man if he were not under the influence of an anæsthetic. A good example of this was a Canadian soldier I saw in August 1916, a man of forty-two, wounded seven hours previously and who had more than two-thirds of his small gut outside his abdomen. The note which accompanied him from the medical officer up the line showed that this

condition had been present for at least four hours; yet this man's condition was quite good, there was slight pallor, but the systolic blood-pressure was 142 mm., and he recovered.

From such data as these it is fair to argue that, although

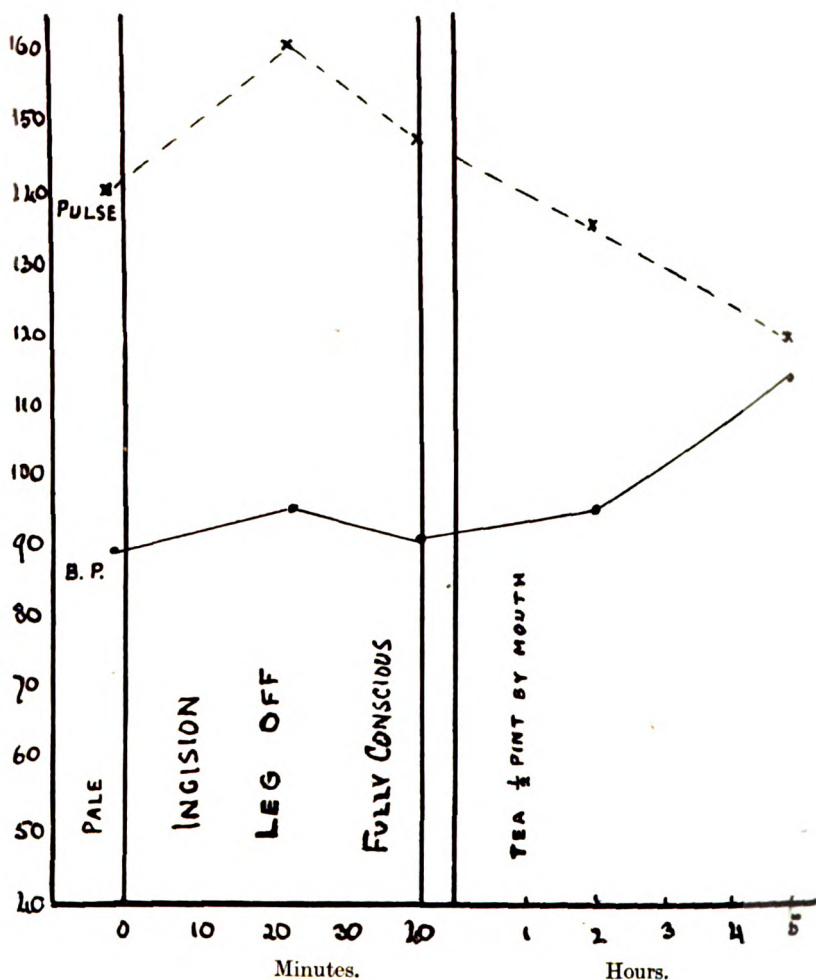


CHART VII. NITROUS OXIDE AND OXYGEN ANÆSTHESIA. Shell Wound Thigh, 22/9/16. Amputation upper  $\frac{1}{3}$  Thigh. Recovered.

ether or chloroform do not in themselves greatly lower blood-pressure, they do render a patient more susceptible to the effects of a certain type of trauma.

There are other methods of producing "operation shock" in man besides the exposure of abdominal viscera; of these none is more effective than amputation of a limb close to the

trunk. Amputation through the upper half of the thigh under ether or chloroform anæsthesia proved fatal in more than 50 per cent. of our cases in France. When chloroform is administered for this operation, the blood-pressure commences to fall during the administration and continues to do so for some hours after, as in the fatal case illustrated in Chart V. With ether, on the other hand, blood-pressure may actually rise during operation, but will fall to a dangerous level within the next two hours (Chart VI). It is this latent period between the end of the administration and the onset of shock which has delayed recognition of the evil effects of ether anæsthesia. There is another drug used for production of general anæsthesia which does not have this disadvantage. If a mixture of nitrous oxide with oxygen be administered to a patient with a concentration of the former just sufficient to abolish consciousness, the most severe operations, including amputation through the upper half of the thigh, may be performed without producing any but trivial changes in his blood-pressure and pulse-rate. Clinically there will be no signs of shock either during or after operation (Chart VII).

#### DISCUSSION

Between 1916 and 1919 much research was devoted to the elucidation of the nature and causation of surgical shock. It is now generally accepted that the lowered blood-pressure found in this condition results from what Cannon<sup>2</sup> has termed "exæmia," by which term he means a diminution in volume of the circulatory blood. According to his theory this diminution is due to stasis of a considerable portion of the patient's blood in dilated superficial capillaries. Dale and Laidlaw<sup>5</sup> have shown that a condition resembling surgical shock can be produced experimentally by injections of histamine, and Bayliss<sup>1</sup> suggests that shock is due to the formation of a substance resembling histamine in tissues subjected to trauma, and that this substance after passing into the circulation acts upon the walls of the capillaries, either abolishing their tone or rendering them pervious to the passage of blood-plasma.

These theories may help us to explain why certain operations performed under ether or chloroform anæsthesia caused lowering of the blood-pressure, whereas similar operations have no such effect when nitrous oxide and oxygen anæsthesia is employed. Dale<sup>4</sup> has found that "shock" is produced when histamine is injected into animals under chloroform or ether anæsthesia, but that there is little lowering of the blood-pressure



when the injection is made during the administration of nitrous oxide and oxygen. It would seem, therefore, that the phenomena are due to some chemical reaction, and the ill effects of ether and chloroform may be due to their entering into combination with some substance set free from damaged tissues and forming compounds which act as toxins to the capillary endothelium. It would not be unreasonable to suggest that nitrous oxide, being a more inert substance chemically, either would not take part in such a reaction, or, if it did, would not form so potent a toxin, and for this reason it has proved the least harmful of drugs used for the production of general anæsthesia.

The deductions to be drawn as regards civil practice would seem to be firstly that chloroform and ether are absolutely contraindicated in operations on patients suffering from traumatic shock, *e.g.* severe "accident cases." The same contra-indication of course holds in abdominal operations such as that for intussusception in children. Perhaps the dreadful mortality of perforated typhoid ulcer might be reduced if gas-oxygen were the routine anæsthetic when these cases were subjected to operation.

We see that chloroform and ether have a profound effect on the condition of the patient in certain types of operation, and it is shameful that these drugs should still be administered as a routine with so little consideration as to the outcome. When these drugs are given, surely the quantity administered should be reduced to a minimum. The barbarous drop-bottle should be discarded in favour of the "warm vapour" apparatus.

As regards spinal anæsthesia, this must be avoided where the patient has had a recent hæmorrhage.

#### KEY TO CHARTS

The continuous line with black dots represents systolic blood-pressure in millimetres of mercury. Diastolic pressures are not entered on the charts.

The dotted lines with crosses indicates pulse-rate per minute.

The red dots represent hæmoglobin percentage in capillary blood.

Scale for the above three curves is marked up the left-hand vertical line.

The next vertical line marks the time when administration of an æsthetic is commenced.

The double vertical line on the right indicates the time when the patient leaves the operating table.

Scale of time is shown along the base line. During operation it is marked in minutes, after operation in hours.

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## A NEW TECHNIQUE FOR THE SIGMA REACTION

By MAURICE E. SHAW, M.D., Radcliffe Travelling Fellow, University of Oxford, Medical Registrar, Guy's Hospital.

THE work here described was carried out in the laboratories of the Clinique Médicale, Hôpital Cochin, Paris, in the service of Professor Widal, and the writer wishes to take the earliest opportunity to acknowledge his deep indebtedness both to Professor Widal and to M. Joltrain, Chef de Laboratoire, for their unfailing kindness and assistance in providing all that was required in the way of laboratory accommodation and apparatus.

In the description that follows a knowledge of the sigma reaction as described by Dreyer and Ward in the Medical Research Council Special Report series, No. 78, "The Serum Diagnosis of Syphilis," is assumed. All references are to the M.R.C. report and not to the original article which appeared in the *Lancet* in 1921.

### TECHNIQUE

The method is based upon the principle of making serial dilutions by means of the "rhéomètre" of Vernes.\* This instrument (Fig. 1) is an automatic pipette made in the form of

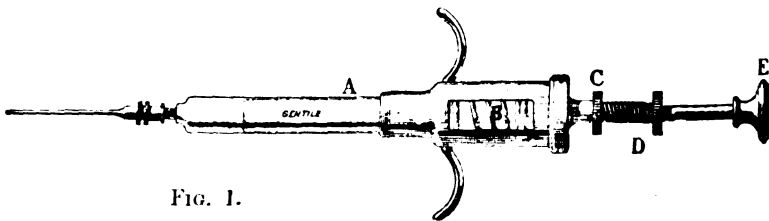


FIG. 1.

a syringe, but provided with a spring (B) to withdraw the piston, and a screw (D) and locking nut (C), by means of which the rest position of the piston and consequently the quantity of liquid delivered can be very accurately regulated. Three of these instruments are required and must be regulated to deliver .2, .4 and .6 c.c. respectively. The first should be labelled "a suspension," the second "serum-saline" and the third " $\beta$

\* These may be obtained from Gentile, 40, rue Saint-André-des-Arts, Paris, and from Etabls. Leune, 28<sup>bis</sup> rue du Cardinal-Lemoine, Paris, V<sup>e</sup>.

suspension." The remainder of the apparatus is precisely the same as described by Dreyer and Ward, except that no dropping pipette is required. All the preliminary steps as described by them in pp. 72-77 are the same, the only difference being in the manner of distributing the reagents (p. 77). This is done as follows :

The agglutination stand and tubes are arranged as originally described. Then—

1. With the " $\alpha$  suspension" rheometer put .2 c.c. of the suspension in tube No. 1.
2. With the " $\beta$  suspension" rheometer put .6 c.c. of the suspension in all the remaining tubes.
3. Next take the "serum-saline" rheometer (.4 c.c.) and put .8 c.c. of serum in tube No. 1 and .4 c.c. of serum in tube No. 2. Then take the dilution tube from its place in the stand (between tubes 5 and 6) and hold it in the left hand. With the rheometer (held, of course, in the right hand) put .4 c.c. of serum into this tube and add .4 c.c. of sterile normal saline. Withdraw .4 c.c. of the mixture and put it into tube No. 3. Add .4 c.c. of saline to the remains of the serum-saline mixture in the dilution tube. Withdraw .4 c.c. of this new mixture and put it into tube No. 4. Repeat this procedure until all the remaining tubes are filled with .4 c.c. of progressively decreasing dilutions of serum in saline.

The test is now set up and can be placed in the water-bath at 37°.

Before passing on to discuss the calculation of the unit content from the dilutions above set out, it will be well perhaps to emphasise certain details of the technique to which attention should be paid.

The rheometers should be clean and dry before starting. This is best ensured by thorough washing with distilled water followed by drying in the oven. The suspensions will, of course, be completely distributed before passing on to the serum and saline. As one is dealing with known absolute quantities of these reagents it is easy to calculate how much of each will be required. .2 c.c. of  $\alpha$  suspension is required for each serum to be tested. Therefore the 10.7 c.c., which is the minimum that can be prepared (p. 74, M.R.C. Report), is sufficient for fifty-three tests. Similarly, 35 c.c. of  $\beta$  suspension furnishes enough for fifty-eight tubes, or seven tests where nine tubes are used.

The first liquid put into the tubes from a rheometer is apt to splash out if directed vertically downwards. It is best, therefore, to direct it against the side of the tube. This only applies to the suspensions.

In making the serum-saline dilutions the rheometer should be washed in saline in between each dilution. After putting the .4 c.c. of serum into the dilution tube the rheometer must be thoroughly washed in saline in order to clear it of all traces of serum. This is quickly done by rejecting about ten fills of saline and using, say, the eleventh to make the first dilution. In making subsequent dilution it is sufficient to reject about five fills of saline before adding one to the serum-saline mixture in the dilution tube. As a matter of fact the amount of washing required gets progressively smaller as the dilutions increase. The rejection of one fill should be sufficient in between tubes 8 and 9. It should be noted that the rheometer always contains a few small bubbles of air (in the case of pure serum or strong concentrations a little foam). This is of no importance, as it is a constant quantity. A rheometer, once well regulated, always delivers exactly the same amount.

In passing from one serum to the next, care must be taken to wash out all traces of the previous serum. This is not difficult, as there is only the tiniest trace of a  $1/320$  dilution left. A few wash-throughs with saline will clear this away. There will then be a small trace of saline left adhering to the glass of the barrel. A few backward and forward movements of the piston will expel all but the minutest trace of this, but as an extra precaution the rheometer can be further washed with a few drops of the serum next to the used.

In distributing the serum-saline dilutions the liquid should be expelled fairly forcibly on to the surface of the suspension already in the tube. In this way shaking each individual tube (a laborious and time-consuming operation) is avoided. The same remark applies to the distribution of the pure serum into tubes 1 and 2, but in tube 1, owing to the small volume of a suspension, the resulting mixture is apt to be imperfect. A glance at the No. 1 tubes will reveal at once whether the serum and saline are well mixed. If not, these tubes must be shaken.

#### CALCULATION OF UNIT CONTENT

No new description is required of the method of reading the degree of flocculation, but, inasmuch as the dilutions obtained by the method above described are not identical with those given by Dreyer in Table I (p. 77) of this paper, some modification of the tables becomes necessary.

It will have been noticed that after distributing the three reagents as detailed above, each tube contains exactly 1 c.c. This is slightly more than the 25 drops provided for by the

original technique, but the reagents are in exactly the same proportions. The definition of a standard  $\Sigma$  unit requires that the volume of serum-saline mixture shall be to the total volume as 1 is to 2.5. By the new technique this proportion is preserved, as the serum-saline mixture is always .4 c.c. and the total volume always 1 c.c. Therefore if one knows the total dilution in which the serum acts in each tube it is possible to calculate the unit content per c.c. of the serum according to the rules laid down by Dreyer on p. 80. These dilutions are shown

TABLE I.

Tube.	Total dilution in which serum acts.	Tube.	Total dilution in which serum acts.
1	1/1.25	6	1/40
2	1/2.5	7	1/80
3	1/5	8	1/160
4	1/10	9	1/320
5	1/20	(10)	(1/640)

in Table I, which should be compared with Dreyer's Table I, p. 77. It will be remarked that the dilutions here given are not identical with those given by Dreyer, but that, while having the advantage of all being whole numbers (except the first two), they increase less rapidly than those in Dreyer's series. Any given figure of the above series is exactly double the previous one. This is not so in Dreyer's series for two reasons. Firstly, because of the variability in the volumes of drops of the different reagents which necessitated a special calculation based upon the relative volumes of these drops; and secondly, because it is impossible, starting with 20 drops of serum, to halve the quantity of serum (or 1/20 dilution of serum) progressively. After putting 5 drops into tube 3 it is necessary to divide this quantity not by 2 but by 2.5, for the next tube (4), which contains 2 drops. Subsequently the halving process continues as far as tube 7, but tube 8 contains 1/2.5 and not 1/2 of the quantity in tube 7. One result of this is that Dreyer's tube 9 has a dilution of over 1/500, while the new technique gives tube 9 only 1/320. It is for this reason that Table I above provides for a tenth tube (which is no extra trouble to set up). The range of the ten tubes will then cover the dilution range of Dreyer's series.

The more even increase in dilution which the use of the rheometer makes possible is no disadvantage. In Dreyer's series of dilutions there is clearly a greater chance of standard flocculation occurring in between tubes 3 and 4 or 7 and 8 than

in between others, so that the chances of standard flocculation occurring actually *in* one of the tubes are slightly greater in the more evenly increasing dilutions of the series shown in Table I above. Another advantage is the simplicity with which such a table as Dreyer's Table III (p. 82) can be constructed. It is only necessary to use Dreyer's interpolation figures to calculate the figures for tube 1, and the figure for each succeeding

TABLE II.

	1	2	3	4	5	6	7	8	9	10
t	1.60	3.20	6.40	12.8	25.6	51.3	102	205	410	820
t—	1.29	2.58	5.16	10.3	20.6	41.2	82.5	165	330	660
s +	1.01	2.02	4.04	8.08	16.16	32.3	64.6	129	258	517
s	.80	1.60	3.20	6.40	12.8	25.6	51.3	102	205	410
s—	.67	1.34	2.68	5.36	10.7	21.4	42.8	85.7	171	343
tr +	.57	1.14	2.27	4.54	9.08	18.2	36.3	72.6	143	286
tr	.48	.96	1.92	3.85	7.70	15.4	30.8	61.6	123	246
tr—	.42	.83	1.66	3.33	6.66	13.3	26.6	53.3	106	213
tr ?	.33	.66	1.32	2.64	5.28	10.6	21.2	42.4	84.8	169

tube will always be exactly double the preceding one. Table II above is merely a modification of Dreyer's Table III (p. 82) calculated from the dilutions in Table I above. It is therefore only to be used in cases of 20–22 hours' incubation, and necessitates the employment of the suspension factor as usual.

By following the instructions given above it should be possible for anyone familiar with Dreyer and Ward's original paper (M.R.C. Report) to put up a nine or ten tube test by the new technique and to calculate the unit content of any serum by making use of Table II above. For routine purposes the details so far given should suffice, but it seems worth while to devote a little space to a description of the possible modifications to which this technique lends itself. For one of the advantages claimed for it is its elasticity, which makes it possible to utilise an almost infinite series of dilutions, of which the series given in Table I is the simplest.

#### MODIFICATIONS OF THE TECHNIQUE

The reason why the particular series figured in Table I was chosen is that it is the nearest approach possible to the series given by Dreyer. But it is just as simple to put up a series of dilutions which will increase very much less rapidly. In making the series described above, equal quantities of serum and saline were mixed and the rheometer was set to deliver a volume equal to the quantity of serum (or saline) used. This

quantity was .4 c.c. By altering any or all of these volumes an entirely different series of dilutions will be obtained. But, in order to be able to calculate the unit content of a serum, not only must the dilution in each tube be known, but the quantity of serum-saline mixture must always be the same. This quantity is taken as .4 c.c. because, when .6 c.c. of suspension are added, the total quantity (1 c.c.) is very nearly equal to the 25 drops of the Dreyer technique, and is about as much as the standard agglutination tubes will conveniently hold. This volume (.4 c.c.), therefore, cannot be altered, and whatever series of dilutions it is proposed to make, a rheometer regulated to .4 c.c. must be used. But the initial quantities of serum and saline put into the dilution tube can be altered at will, and any alteration will modify the resulting dilutions. For example, if, instead of taking equal quantities of serum and saline (or .4 c.c. of each), one starts with a stronger concentration of serum by mixing .8 c.c. of serum with .4 c.c. of saline, a more slowly increasing series of dilutions will result. It must be borne in mind that the modification will only affect the tubes in which the serum is diluted in saline. The first two tubes contain no saline, and the dilutions cannot, therefore, be modified. In calculating a new series of dilutions (by a method shortly to be described) one must start always with the third tube.

The dilutions resulting from any such alterations of the relative volumes as is here indicated will depend upon two factors :

1. The dilution of serum in the dilution tube before starting the distribution. This will always be referred to as the "initial dilution."
2. The ratio  $\frac{\text{total volume in dilution tube}}{\text{volume delivered by rheometer}}$

Now for the purposes of this test it has been seen that the rheometer must always deliver the same volume (.4 c.c.). Therefore it is only possible to vary the initial dilution and the total volume in the dilution tube. This latter volume remains a constant quantity, as the amount of saline-serum removed at each operation is replaced by an equal quantity of saline.

In order to calculate the actual dilutions in any series, a formula has been constructed to cover all possible alterations of the three potential variable quantities. This formula is :

$$\frac{I}{(x - a)^{n-1}}$$

where  $x$  = total volume in dilution tube  
 $a$  = volume delivered by rheometer  
 $1$  = initial dilution  
 $y$   
 $n$  = number of tube in series.

(Note :— $n$  refers to the tubes in the series of serum-saline dilutions only. The first tube of this series is the third tube of the completely set up test.)

It is obvious that the first dilution equals the initial dilution, since .4 c.c. is simply removed from the dilution tube and put into the first tube of the serum-saline series (third tube of the complete test), and this dilution must be known. To calculate succeeding dilutions it is only necessary to multiply the figure for the preceding one by the expression  $\frac{x}{x-a}$ . This will give the dilution of *serum in saline*. To obtain the dilution of serum in saline + suspension this figure must be multiplied by 2.5. The rapidity of increase of successive dilutions will be inversely proportional to the ratio  $\frac{x}{a}$ . In the series figured in Table I above  $x = a = .4$  c.c., so that  $\frac{x}{a} = 1$ . The only question, as far as technique is concerned, which remains to be considered is the best method of increasing the value  $\frac{x}{a}$  and so ensuring a more gradual increase in dilution. The simplest procedure is to make use of the .4 c.c. rheometer and to mix .4 c.c. of saline with some multiple of .4 c.c. of serum. By this means values of 2, 3, 4, and so on, are obtained for  $\frac{x}{a}$ . The disadvantage is that a fairly large amount of serum may be required, and this is not always available. But the initial dilution may just as well be made with an ordinary pipette graduated to .01 c.c. In this way the quantity of serum available can be adapted to the series of dilutions required. But the greater the value of  $\frac{x}{a}$  that is required, the larger, generally speaking, must be the quantity of serum. This is due to the necessity of having  $x$  sufficiently large to be workable ( $x$  must clearly be greater than  $a$ , and should be certainly not less than  $1.5a$  where  $a = .4$  c.c.), and to the fact that a high concentration (or low dilution) of serum is usually required for the initial dilution—i.e. greater than  $1/2$ . If  $y = 2$ , the total dilution, after addition of suspension, in tube 1 of the serum-saline series (tube 3 of test), will be  $1/5$ , which

is the same as in tube 3 of Table I. In order to ensure having a tube in between the dilutions of tubes 2 and 3 of Table I, it will be necessary that  $y$  be less than 2, and in order to secure less rapidly increasing dilutions  $\frac{x}{a}$  must be greater than 1. By bearing these facts in mind the technique can be adapted to provide any sort of dilution series required.

## EXPERIMENTAL RESULTS

As this technique was elaborated while using the  $\Sigma$  reaction for other purposes it has not been possible to make a very large number of experimental observations. Only a limited number of the available sera could be used, as, in order to compare the results of two techniques, a double quantity of serum was necessary. Further, it was thought better to select those sera which were expected to give a positive reading.

Thirty-five different sera and three different series of dilutions were used, though in all cases except two the test was also put up in the usual way (with the dropping pipette). In twenty-four cases the table of dilutions shown in Table I was made use of. This series is called  $\Sigma a$  to distinguish it from the  $\Sigma$  series (Dreyer's Table I). The two other series used are called  $\Sigma ai$

TABLE III.

No. of tube.	$\Sigma a$ . Serum 1. Saline 1.		$\Sigma ai$ . Serum 2. Saline 1.		$\Sigma aii$ . Serum 3. Saline 1.	
	Dilution in saline.	Total dilution.	Dilution in saline.	Total dilution.	Dilution in saline.	Total dilution.
1 (3)	1/2	1/5	1/1.5	1/3.75	1/1.33	1/3.32
2 (4)	1/4	1/10	1/2.25	1/5.63	1/1.78	1/4.45
3 (5)	1/8	1/20	1/3.38	1/8.45	1/2.37	1/5.93
4 (6)	1/16	1/40	1/5.06	1/12.7	1/3.16	1/7.90
5 (7)	1/32	1/80	1/7.59	1/19.0	1/4.21	1/10.5
6 (8)	1/64	1/160	1/11.4	1/28.5	1/5.62	1/14.1
7 (9)	1/128	1/320	1/17.1	1/42.7	1/7.49	1/18.7
8 (10)	1/256	1/640	1/25.6	1/63.7	1/9.99	1/24.9
9 (11)			1/38.4	1/96.1	1/13.3	1/33.3
10 (12)			1/57.7	1/143	1/17.8	1/44.4
11 (13)			1/86.5	1/216	1/23.7	1/59.2
12 (14)			1/130	1/324	1/31.6	1/79
13 (15)			1/195	1/487	1/42.1	1/105
14 (16)			1/292	1/730	1/56.1	1/140
15 (17)					1/74.8	1/187
16 (18)					1/99.7	1/249
17 (19)					1/133	1/333
18 (20)					1/177	1/443
19 (21)					1/237	1/593



and  $\Sigma a_{ii}$ . The dilutions of all three series are seen side by side in Table III. At the head of each column the relative quantities of serum and saline used to make the initial dilution are shown. In all cases the unit was .4 c.c., as the initial dilutions were made with the serum-saline rheometer. The dilutions before and after addition of suspension are shown, and in each case the series are carried to a dilution higher than 1/500.

TABLE IV.

No. of serum.	$\Sigma$ .	$\Sigma a$ .	$\Sigma a_i$ .	$\Sigma a_{ii}$ .	No. of serum.	$\Sigma$ .	$\Sigma a$ .	$\Sigma a_i$ .	$\Sigma a_{ii}$ .
35	8.12	7.89			59	0			0
36	8.16	7.89			60	2.40	2.70		
37	13.4	12.8			61	4.80	4.10		
38	108	102			62	4.10		3.84	
39	8.32	8.08			63	3.50			2.41
42	102	107			64	3.90	4.10		
45	76.6	81.6			65	3.10			2.90
46	16.6	13.7			67	3.0			3.40
47	3.50	5.12			68	0		0	
48	3.0	3.6			69	0	0		
49	2.70	2.60			70	4.40	4.70		
50	5.0	8.40			71	7.80	6.80		
51	4.10	5.12			72	7.0	5.40		
52	1.94	2.10			73	1.70	1.80		
53		5.12	5.60		74	2.40	3.60		
56			46.5	59.3	75	3.20			4.70
57	0		0		76	7.0		5.90	
58	0		0						

The results are tabulated in Table IV, and call for little comment. The sera numbered 46, 50 and 72 are the only ones in which the figures obtained by the ordinary  $\Sigma$  technique are seriously challenged by the  $\Sigma a$  technique. In such cases the writer feels that the  $\Sigma a$  reading has as much, if not more, claim to be considered accurate as the  $\Sigma$  reading. Any error inherent in the new technique described must be constant, and the closeness of the majority of the readings in Table IV make such error, if it exists, negligible.

## SUMMARY

1. A new technique for Dreyer and Ward's Sigma reaction is described which renders that test more simple and quick to carry out and gives results of equal if not greater accuracy.

2. Further, this technique is capable of infinite variations which may be useful to workers who are not using the test merely for routine purposes.

3. The principle of making dilutions with the rheometer (for which no originality is claimed) is described in some detail, as it is applicable to many branches of medical laboratory work.

4. The results of a comparison of the new and old technique on thirty-five different sera are given.

In conclusion the writer wishes to thank Professor Dreyer and his assistants in the Department of Pathology in the University of Oxford for providing him with the antigen used in the experimental work, for opportunities of seeing the details of the technique as carried out in Oxford, and for help with certain theoretical points concerned with it.

## A CASE OF ANÆMIA SECONDARY TO DENTAL SEPSIS

By G. G. EXNER, L.D.S., and CYRUS IVE, M.B.  
(From the Medico-Neurological Clinic, Guy's Hospital.)

THE patient was admitted into Mary Ward under Dr. G. H. Hunt in August 1924. She was a woman of 34 years of age, married but nulliparous, living in the country, and under good conditions. She gave a good family history, and this was the first illness that she had experienced.

For six months prior to admission the patient had noticed a feeling of languor, shortness of breath and palpitations. She had also had several attacks of epistaxis, and had noticed that her menstrual losses were increasing. She got steadily worse, so she consulted her doctor, who sent her to bed. During this time she had an irregular pyrexia, the temperature rising to 105° F. on one occasion. She never complained of any gastrointestinal upset, of soreness of the tongue, or of pins and needles or numbness of the extremities.

On admission she was obviously very anæmic. Her skin was a muddy, but not lemon-yellow, colour. The temperature was raised to 100° F., the pulse-rate regular at 110 beats per minute, and the respiration rate 20. The respiratory, cardiovascular, alimentary, genito-urinary, and nervous systems presented no abnormality to physical examination. Special investigations were made, therefore, to assist in the differential diagnosis. The diseases particularly considered were infective endocarditis, septicæmia, Addison's anæmia, and anæmia secondary to some source of sepsis.

*Examination of the blood.*—Red corpuscle count 1,525,000 per cub. mm.; Hb. 21 per cent.; colour-index 0·7. Some anisocytosis, but no megalocytosis; no nucleated red cells.

White corpuscles count 1,500 per cub. mm., with relative lymphocytosis.

Two blood cultures, done by the "dilution method," were negative.

Van der Bergh's reaction; direct negative; indirect a slight and slow reaction. This reaction therefore showed excessive hæmolysis.

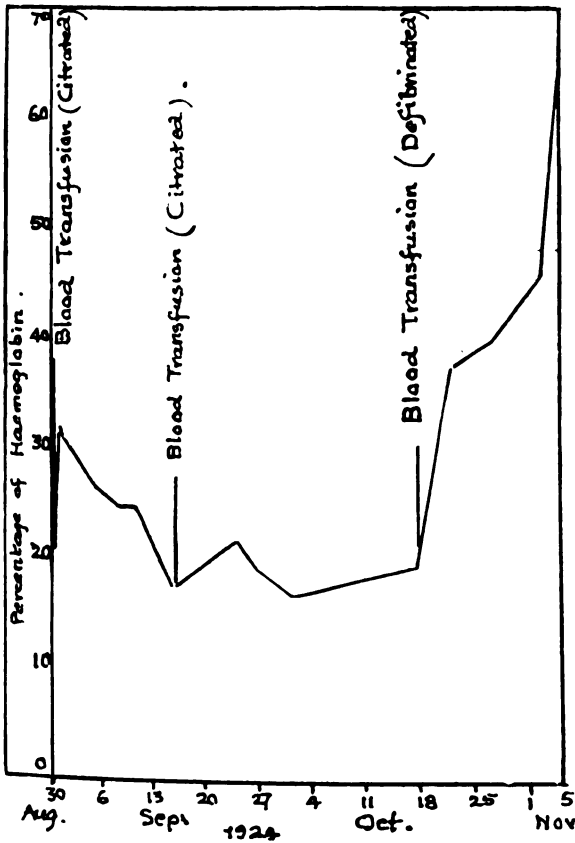
Wassermann reaction negative.

Fractional gastric test-meal : curve was normal.

Teeth standing were  $\begin{smallmatrix} 87 & 21 & 12 & 78- \\ 54 & 321 & 123 & 5 \end{smallmatrix}$ . Marked absorption was apparent around lower incisor and upper molars; extensive pockets from which very little pus or exudate could be forced.

From the investigations we drew the following conclusions.

(1) The diagnosis was unlikely to be infective endocarditis



or septicæmia, because of the absence of cardiac signs, of positive blood cultures, and of leucocytosis. (2) The diagnosis was certainly not Addison's anæmia, as there were no symptoms other than those due to the anæmia, the blood picture was definitely not that of Addison's anæmia, and the fractional test-meal gave a normal hydrochloric acid curve. The blood picture in Addison's anæmia shows anisocytosis with increase in the average size of the red cells, of the nucleated red cells, and megaloblasts, and generally a high colour index. We

therefore concluded that the anæmia was hæmolytic, though not of the Addisonian type, and was entirely due to dental sepsis. The main object in treatment was therefore to eradicate the dental infection. Obviously we had to proceed carefully, for the patient's general condition was extremely bad. Immediately after admission a blood transfusion was performed, which had the effect of raising the hæmoglobin to 33 per cent. Citrated blood was used, and there was a considerable reaction. Two teeth were then extracted under gas. Cultures were taken from the apices and swabs from the pockets. Cultivation gave a growth of *streptococcus longus*.

Profuse bleeding occurred from these extractions, which was ultimately arrested by tight plugs of oil of turpentine.

The following day another blood transfusion was given.

The rest of the teeth with the exception of  $\frac{21}{8} \frac{12}{8}$  were removed at the rate of one per week. A local anæsthetic containing adrenalin was used, and the sockets plugged immediately after extraction. Inoculated media from all these teeth gave a growth of *streptococcus longus* of a definite hæmolytic type.

A vaccine was prepared and given. Seven weeks after admission, eight teeth having been removed and two blood transfusions given, the hæmoglobin percentage was still only 18. It was therefore considered advisable to give another blood transfusion, and on this occasion defibrinated blood was used from a donor, who four hours previously had been inoculated with 50 million dead streptococci cultivated from the patient's teeth. The effect of this transfusion was dramatic; the pulse-rate fell from 100 to 80 and the Hb. rose to 40 per cent. very rapidly. It has risen steadily ever since, and the patient has now been discharged fit. The blood picture at the time of discharge was R.B.C. 4,400,000 per cub. mm.; Hb. 64 per cent.; colour-index 0.73; white count 6,000 per cub. mm. with marked lymphocytosis.

We thought the success of the treatment depended on—

(1) Gradual extraction of teeth for removal of all infective foci.

(2) Early administration of an autogenous vaccine.

(3) The use of defibrinated blood following the injection of the patient's vaccine into the donor instead of citrated blood.

Our thanks are due to Dr. Hunt and Mr. Pearce for permission to publish this case.

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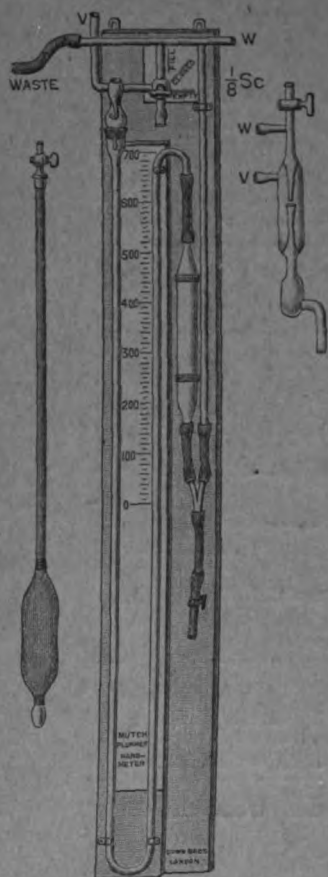
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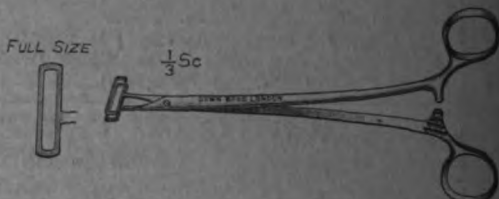
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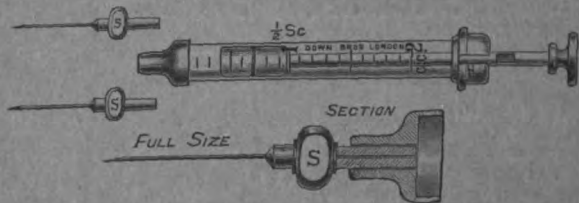
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*W. H. A. Jacobson*

W. H. A. JACOBSON, M.A., M.Ch., Oxon., F.R.C.S.,  
Surgeon to Guy's Hospital, 1876 to 1905.

## IN MEMORIAM

WALTER HAMILTON ACLAND JACOBSON,  
M.CH. OXON, F.R.C.S.,

CONSULTING SURGEON TO GUY'S HOSPITAL.

By R. P. ROWLANDS, M.S., Surgeon to Guy's Hospital.

WALTER HAMILTON ACLAND JACOBSON, who was born at the Little Red Brick House, New College Lane, Oxford, in March 1847, died at Lordine Court, Ewhurst, Sussex, on September 16, 1924, aged 77. He was the second son and the sixth child of the Rev. William Jacobson, late Scholar of Lincoln and Fellow of Exeter, then Public Orator of the University and Vice-Principal of Magdalen Hall, and soon afterwards Regius Professor of Divinity. Eighteen years later he was consecrated Bishop of Chester—"the single-minded Bishop," as Dean Burgon aptly called him in his *Lives of Twelve Good Men*. The "learned, faithful and pious Bishop" died of cancer in 1884. His wife was the youngest of the six charming and accomplished daughters of Dawson Turner, the banker, of Great Yarmouth, friend and collaborator of John Sell Cotman, the antiquarian. Her eldest sister married Sir William Hooker, the great botanist, and another married Sir Francis Palgrave, the historian. Walter Jacobson was the godson and namesake of Sir Henry Acland, then Regius Professor of Medicine, the father of the study of natural science at the University. Thus, from his earliest years, he lived in the atmosphere of learning: little wonder that he became such a good classical scholar. Of the Bishop's ten children only one, a daughter, survives Jacobson.

Being considered a delicate boy, Walter was sent to a preparatory school at Exmouth "to be in the sea air." He always spoke of the Headmaster, Mr. Penrose, with great respect. He went on to Winchester and later to Corpus Christi College, Oxford, where he took a second class in Moderations and graduated with first-class honours in the School of Natural Science in 1869. Soon afterwards he entered and rapidly made his mark at Guy's Hospital Medical School; he qualified as M.R.C.S. in 1872 and became F.R.C.S. in 1875. Twelve years later, when the M.Ch. degree was established at Oxford, he

believed it to be his duty to sit for this higher examination before he could conscientiously examine others in it. This was characteristic of his attitude of mind. He was the first to take the degree—in March 1887.

In course of time he became demonstrator of anatomy, assistant surgeon (1876), teacher of operative surgery, and full surgeon (1900) at Guy's Hospital, and he rapidly developed into a wonderful and unrivalled teacher of anatomy and surgery. He was assistant surgeon for over twenty-four years, full surgeon for five and consulting surgeon for nearly twenty. He was also surgeon to the Hospital for Women and Children in the Waterloo Road, where he gained much of his earlier experience as an operator.

From 1893 to 1898 he was an examiner in anatomy, and from 1900 to 1905 a member of the Court of Examiners in Surgery for the Royal College of Surgeons of England, an appointment which he conscientiously resigned when he gave up his work at Guy's. He also examined in surgery for the University of Oxford. He was a fair and sympathetic examiner, although some candidates were afraid of him.

No man ever gave more devoted and loyal service to his Medical School and Hospital; his work at Guy's came before everything else. During "take-in" or when he was "in Clinical" he was often in the wards by 7 a.m., even when he had been operating late at night. Then and at other times he frequently walked to the hospital from Great Cumberland Place, a distance of about four and a half miles. His lectures on anatomy were extremely popular, for he had the gift of making the "dry bones live": his apt surgical allusions were designed to show the great importance of anatomy in practical surgery. He prepared these and his surgical lectures with the greatest care, and he generally arrived a long time beforehand and covered several blackboards with beautifully written, accurate notes, and excellent drawings in coloured chalk. This work was often done very late at night, Jacobson afterwards retiring to rest at the Bridge House Hotel. He often distributed printed notes of his clinical lectures which men treasured for many years. His classes, surgical lectures and clinical demonstrations were crowded, for his brilliant intellect, extensive knowledge, fascinating personality and scintillating wit made everything he said interesting and memorable. Men frequented his lectures not only to gather sound knowledge and to be "signed up," but also to obtain mental refreshment and stimulation. Like the great teacher he was, he took pains to make everything clear to the duller member of his class.

He kept every member awake, alert and even anxious, because he asked the most searching questions at random. No one knew when his turn would come or how his answer would be received: it was always without favour, often with pity or scorn, sometimes with praise. He believed that this was the best method of teaching and the only way of finding out what the men did not know and of keeping his teaching at the proper level. He frequently attended the examinations and made notes of the questions asked by the examiners and the answers expected, so that he might make his teaching more effective. No wonder that few who had attended his classes ever failed in their examinations. For over twenty years his out-patients were most instructive and amusing: they were packed with students to the last.

His kindness, sympathy and generosity to his hospital patients were so extraordinary that many walked long distances to see him and to show themselves years after they had completely recovered. Many a rogue did not go away empty-handed. Jacobson was pleased to demonstrate and criticise the ultimate results of his operations, to the great advantage of his class.

His love for the students was deep and genuine, he advised and helped them in all sorts of ways without thought of reward. "Pom" 's personal ascendancy over his pupils was so remarkable that he could say what he liked to them. He told them the blunt truth and exercised his ready wit and sarcasm upon them, and he was sometimes hasty and unjust, but he never meant any harm. Some resented his scathing sarcasm, but most men realised that it was all for their good, and they loved and feared him in return: some were a little hurt because he appeared to neglect them in class, but "Pom" knew full well that these did not need any special attention. He did not like the man who made excuses or sulked when "spoken to," but loved the heroic fellow who took his punishment smiling.

Jacobson's boundless energy, classical as well as technical knowledge, and great literary ability enabled him to write his most enduring and laborious work, the *Operations of Surgery*. Afflicted as he was for many years with severe insomnia, he made good use of the time it afforded him, and did most of his writing, standing at his desk, after waking in the small hours of the morning. This book was the outcome of a strong belief which he "had held for many years, that a work on operative surgery, which aimed at being more comprehensive in scope and fuller in detail than those already published, would be of service to many who had recently been elected to

hospital appointments, and to those who were working for the higher examinations." The first edition of this book appeared in 1888 and succeeded beyond his expectations, for its wealth of clinical experience, its accurate, lively and critical descriptions of operations, its sound common sense and fascinating style made it indispensable to every surgeon. Not a few still speak of it as their Bible. It was the first really satisfactory book written in English on general operative surgery, giving accurate information and sound advice, based on exhaustive researches into the literature of the subject and controlled by personal experience, absolute honesty and sound judgment. Since then the book has run into six editions.

Jacobson wrote another good book on *Diseases of the Male Generative Organs*, published in 1893, and he revised several editions of John Hilton's great work, *Rest and Pain*. His contributions to the *Guy's Hospital Reports*, of which he was joint editor for many years, were numerous and valuable, lucid and charming in style. Notable among these are his "In Memoriam" notice of John Hilton and his account of "Five Cases of Chancre in Medical Men." He also wrote some of the best articles for Holmes' *System of Surgery* and for Heath's *Dictionary of Surgery*. He was the English collaborating editor of the *Annals of Surgery* from 1896 to 1903, when he retired from private consulting practice, chiefly on account of ill-health. He also needed more time to revise the fifth edition of his *Operations of Surgery*, which appeared in 1905. Through his writings Jacobson became known all over the world, and many medical men came to see him and his work at Guy's, but, truth to tell, Jacobson was rather disappointing as a practical surgeon.

It was a pity that he had to wait so long before "getting his beds," for this interfered with his technical training as an operating surgeon. He knew well that surgery is an art which is best learnt in youth, although the wisdom of years contributes to the making of a master. He often complained that he had not had enough operating in his early years as Assistant Surgeon, but this was chiefly because he lived away in Finsbury Square and had to be fetched in a cab when required, whereas his senior colleague lived in St. Thomas' Street close to the hospital. On studying a list of operations performed by a House Surgeon, he told the latter that he had done more operations in six months than he, Jacobson, had done in seven years as Assistant Surgeon.

Although he appears to have been a delicate boy when he first went to school, Jacobson developed and played football

in the "Commoner" team at Winchester. One of his distinguished contemporaries, Sir Francis Champneys, remembers him as a "quaint and humorous boy, bright and merry." A relation says that at Oxford "he enjoyed rowing and would have been in the Corpus boat when it was head of the river had not his father wished him to see a doctor, who forbade it, fearing heart trouble. . . . He chose his profession very early and never wavered from his choice, but being one of ten children, and having two younger brothers and an invalid sister, he set his mind on beginning his medical studies on leaving Winchester, to get sooner off his father's hands. It was a real effort to him to give up this plan and to go to Oxford at his father's wish." This unselfishness, not to say martyrdom, was typical of Jacobson.

At Oxford he appears to have been "shy and peculiar, the other students stood in awe of him and did not consider him to be like other men." One of his contemporaries says that he "formed the opinion that he was a man of sterling character and very kindly, but that he had not then, at any rate, the gift of sociability, nor had he, I think, much humour. I mentioned, for instance, that I liked 'stinks,' this being the universal term for Natural Science. He held up his hands in horror and implored me never to use such horrid language. He was grave, reserved and easily shocked by what he considered the improprieties of speech and conduct current among undergraduates."

Dr. R. Chicken, one of his fellow-students at Guy's, writes the following interesting account, as he knew him over fifty years ago. "There are not many now living who remember him as a first year's man, and of these none knew him at all intimately, possessing, I should think, only very hazy recollections of what must have been a mere nominal 'acquaintanceship.' All those who then knew him best are gone; I can think of none now living who saw as much of him as I did. Why do I put an inverted comma to mark the word 'acquaintanceship'? Because it had a special meaning in Jacobson's phraseology, the origin of which was this. He was spending an evening with me once in my lodgings in Nelson Square when, in the course of the conversation, I alluded to 'your friend —,' when he, in a moment, said (in his gruffest Jacobean voice): 'Friend. I have *no* friends; acquaintances, if you please, but *no* friends.' Of course this soon became public property and was recognised as a characteristic incident. His position in the everyday life of early studentship was peculiar. He was like no one else: his mannerisms were so entirely his



own that they struck everyone as 'odd.' There was no doubt about their reality, they were evidently a part of himself, but they were always so markedly present that it was impossible to doubt but that they were encouraged rather than suppressed by him. I was told by one that knew the family that they were inherited from his father, the Bishop of Chester, in common more or less also with his brothers. But the brother who had a living Seven Dials way, a fine, big muscular Christian, seemed to have but little of the taint, not sufficient to attract attention even when sought for. I remember seeing him standing up in the Colonnade waiting for W. H. to come up. What a contrast when they met! One a big, smiling, happy-looking Christian; the other an ascetic, austere follower of a severe order of life. Jacobson was liked and was popular because he seemed never to pose for his own profit, or desire to appear superior to others. He seemed always to court humility and place himself in an inferior position to his fellows. In this respect I think I should be justified in saying that some of the effort was artificial, becoming apparent by reason of its exaggerated character. I once, for example, asked him to come down and read a paper before our Medico-Chirurgical Society. He wrote saying that he did not feel sufficiently erudite to do so, as reading a paper before the talented men of — (mentioning the town where I practised) was a very different thing from the same for 'the Galens of Brixton.' But on second thoughts he would consider it, but it would take time. He enclosed the headings of the paper he would put together. This was a close-printed page, of notepaper size, of difficult and abstruse questions in surgery. In the case of any other person it would have been put down as an attempt at pulling my leg, or a ponderous joke; but who could ever conceive such a thing as a joke issuing from those pouting, compressed lips? It would be the biggest joke of all. No, he was quite serious, for he wrote me further on the subject, going nervously deeper into the subject of his paper, until it became so comprehensive that to read the headings only would have taken the usual twenty minutes. It was his natural humility pushed to an unseemly extreme.

"Not only was he liked but he was respected, and the men of his year looked up to him, even then, as their superior. He was also kind-hearted to anyone, even a stranger, who asked his help, and it was never given grudgingly or with the air of a superior, but rather as a fellow-worker anxious to help. I remember once asking him to do something for me. He assented so readily that it had the appearance of discharging an obliga-

tion, for he said that it was I who had first showed him how to put a bandage on. There *may* have been some trifling incident which he recollected when we were Assistant Surgeon's dressers in the O.P. room, but I had no recollection of it. Then his Oxford friend, George Parker of Oriel, a far too popular man—how can anyone forget the way he always funked his 'First College' till Jacobson tackled him and stuck to him like wax until he had got him through? Can anyone think of the greatness of the task? Parker, a man of our own year—always called 'The Colonel'—had been six years at Oxford with Jacobson and six years at Guy's, till I left after my final F.R.C.S., and he had not passed, at either Oxford or London, a single examination! Then, as I said, Jacobson took him up and he passed. But what a task it must have been! What a close continuous effort, for Jacobson, entirely self-imposed. This was a typical incident.

"I don't think that I ever saw Jacobson smile. He may have done so, but he was always far too serious. As he lived longer among other men these peculiarities may have become modified, but in his early years he always remained on his guard lest he should slip away from the line he had vowed to take. His great effort seemed to be to avoid observation, and in later years many were the visits paid to the Guy's Museum at midnight so that there should be no witness of his work. He never pushed himself to the front or endeavoured to draw attention to himself. His voice was rarely heard and then only in low, deep notes, in a few sparing words. There was observable a tendency to blend sarcasm with his remarks on things he despised, and though he might bring himself in close communion with men of inferior metal, it was through the friendship of their necessity, not of heart and feeling: when the necessity was passed the event was over and done with, no friendship remained, only acquaintanceship. It was a unique position to have to work on a given subject or committee with him; one would feel oneself pushed on, but with such protestations of incapacity that one was half inclined to believe them entirely genuine. The basis or motive of his life was sound and genuine, but he allowed emotion to enter rather more into his daily work than men of his mental calibre usually do. He was too nervous in his desire to be correct. Take his book on *Operations* as a type of himself. It professes to be a text-book, but dissatisfaction with facts as stated in the text appears on almost every page, so there are notes to rectify any error and notes to those notes, to fill up any gap, until the mind of the learner is confused and he wonders where truth lies. Instead

of a text-book for students Jacobson has written a finished treatise and commentary for experts, the bane of all text-books for beginners. So with his conversation. He was so afraid to say what he thought was right, for fear of being wrong, that it ended in an explanation or a dubious silence. I am speaking now only of his early student days. Although by men of his time he was looked upon as an oddity, he was thoroughly respected by all as an earnest, hard-working, generous man.

"You will see in this rambling sketch of mine what an earnest respect I had for him, although he seemed to dislike the intimacy which ripened into warmth with men like Fred Taylor and Higgens, and others of the same period."

Sir William Hale-White says of Jacobson: "He was the best teacher I have ever met, partly from a great natural gift, partly from the trouble he took. The theatre was always packed, those who were not there in time could not find a seat. Crowds attended his out-patients, his demonstrations and his visits to the wards. Hilton, in his last years, said, 'I know I used to be down upon the men, but I did it for their good.' The same was true of Jacobson. His straight speech to the idler would have been resented had it come from anyone else, but all forgave on account of their knowledge that his object was to make them learn. No one will ever know a tithe of all that he did for the students when they were in trouble; they loved him and recollected him long after they had left hospital. He was a man who always remembered little things. I was sitting with Jacobson in his study one evening when Sir Henry Howse's servant brought him a brace of pheasants. He went into the hall, thanked the man, gave him some cigars for his trouble, came back and in two minutes jumped up saying that, as Howse did not smoke and disliked the smell, he ought not to have given his servant cigars. Thereupon he rushed down the street, recovered the cigars and gave the man money instead."

One of his pupils writes in the *Guy's Hospital Gazette* (October 25, 1924): "The writer of Mr. Jacobson's obituary notice in *The Times* said very truly that his death would release a flood of memories. The vision that arises before me, undimmed by the lapse of thirty-five years, is of a spare figure with hat tilted slightly backwards, walking with rapid stride along the Colonnade: then the ascetic face and humorous twinkle of the eye. Another picture is in the wards, and who will ever forget the ominous shake of the head and contemplation of his waistcoat buttons (very handsome ones, by the way) with

which he would accompany a grave prognosis? My first introduction to the wards was as his clerk, and the first operation was a removal of the breast. He told me that he was very particular about the accuracy of his reports and that I had better see what I could do and he would correct it next day. His *Operations of Surgery* had just been published, so I went to the Library, got it down and, with necessary minor alterations, 'lifted' the entire operation from the book. Next day it was shown up and, when he had read it, his comment was, 'a most excellent report, sir, I could not have written a better myself,' and to this day I have never satisfied myself whether he knew how literally true that was. However, he always treated me with great kindness then and during a subsequent term as dresser. Any small service, not precisely 'in the bond,' would be sure of recognition, perhaps by a handful of cigars extracted from his pocket or possibly by a book to a non-smoker. If he asked a dresser to watch a case in the evening he would produce money to pay for his dinner, and this the wise accepted without demur. Sometimes a new man who did not know his ways would be inclined to resent it, and nothing annoyed him more. He would have none of it. 'It's your disgusting pride, sir,' he would say. 'You wish to lay me under an obligation.' After that, there was nothing for it but to 'swallow the insult.'

"It was a saying amongst us that however much fault 'the Master' might find with his dressers he would allow no one else to do so. It happened that two of us had tiresome septic cases for weeks in 'Sammy,' and regularly twice a week he abused us for their condition and our scandalous neglect of one of his favourite weapons, the 'sharp spoon' ('Whatever you take with you into practice, don't forget a sharp spoon'). On our last round with him he stopped at these beds and we prepared for a repetition, but to our astonishment he told us that the cases did us the greatest credit and that only our care and attention had saved the limbs from amputation, on which he had once or twice nearly decided. He had his failings, of course, but there was never anyone quite like him, and 'there will be no further reproduction, as the mould has been broken.'"

Another pupil, T. Brown, tells the following amusing incident: "Mr. Jacobson was above all an autocrat, and in the early '90s I remember he took his dressers at times to the Waterloo Hospital for Children and allowed them to operate. On one occasion I myself and a student named D'Albon had this privilege. Mr. Jacobson remarked to D'Albon, 'You are

the surgeon and you are to treat me as the dresser.' On making the first incision D'Albon awaited the sponge, but there was nothing doing, until Mr. Jacobson said, 'You are to give me instructions, for I am the dresser only.' To our consternation, D'Albon, in a low voice, said, 'Sponge, Pom,' and throughout the operation maintained the same voice with 'Spencer-Wells, Pom,' 'ligatures, Pom,' etc. 'Pom,' so taken aback, said



*William H. Foster*

Dr. Jacobson, Bishop of Chester.

nothing, but we had a little dinner that night in Cumberland Place."

In the course of time Jacobson grew to be very like his father in mind and body. He had for his "sainted father, the Bishop," unbounded respect, love and veneration, and he deliberately directed and modelled his life in conformity with what he knew to be his father's wishes and ideals.

When I first knew Jacobson, over thirty years ago, he was Assistant Surgeon and Lecturer on Anatomy and at the height of his fame and power. He presented a distinguished and

striking figure. A little over middle age, his hair was already white and scanty, he was of medium height, very thin, slight and extraordinarily quick and active; his step was peculiarly springy. His forehead was high and his nose prominent, his face lean, ascetic and expressive. He was a great walker and believed in taking plenty of exercise in order to keep himself fit. He was most energetic, restless and brimful of knowledge. Although a judge of the good things of life, he ate very little and was most abstemious in his habits, having mastered all the desires of the flesh by a rigorous training which he seemed to enjoy. He had an infinite capacity for taking pains combined with great natural ability and determination. With all this he was, like the rest of us, very human and apparently full of superficial contradictions, mannerisms and eccentricities. He was a great gentleman, but he prided himself on his blunt manners: unselfish to the verge of selfishness, over-sensitive and quick to resent imaginary insults, he was difficult to deal with, especially as a colleague. He had many grievances which he seemed to cherish; quick to espy an antagonist, he was his own worst enemy. His quick tongue gave offence on all sides, but he himself was most sensitive to ridicule. Generous to a fault in his treatment of others, he treated himself badly. He allowed himself very little leisure for recreation or pleasure. He never went to a theatre until he was over middle age, when he was taken by a friend. He would see only Shakespearian plays, which he carefully read beforehand and thoroughly enjoyed.

He was the hero of numberless tales which are still told wherever Guy's men meet. Self-conscious, shy, proud and austere in manner, he appeared to be somewhat vain, pompous and unapproachable, but in reality he was very modest, unassuming and sympathetic. Once a stranger, misled by his distinguished appearance, mistook him for a famous knight and asked, "Are you Sir X.Y.?" "No, sir," was the reply, "just common clay."

He had a great contempt for anything that sounded false or savoured of self-advertisement, but he was very quick to discover merit and scrupulous in giving credit for good work done. In short, he was a man of unblemished and fine character, high ideals, great attainments, lovable and fascinating personality; somewhat capricious in temper and peculiar in manner, he was not always easy to understand. Too honest to suffer fools gladly, he must have found his ready wit and lack of tact somewhat of a hindrance to him in his private consulting practice, with the result that he did not reap quite

as well as he sowed. But, after all, Jacobson never did anything for worldly gain, but always did what he thought was right. The main motives of his life were Duty, Honour and Love, and he had his reward in the love and affectionate remembrance of many hundreds of students and patients who came under his influence and care. To a student holding a hospital appointment he wrote: "I don't wonder this month with its many worries and petty annoyances has troubled you. There are few greater lessons one can learn than how to meet the small things of life cheerfully, and to walk amongst them with one's head up. Looking back on a life clouded by twenty-four years of Assistant Surgeoncy I see how I failed to learn that lesson."

The following illuminating letter was written during the War to two of his old pupils on active service in France:

"To two old friends: Your letter, with its kindly words, gratified me much and brought back memories ever pleasing and welcome. But you probably know me well enough to understand that such memories cannot but be tinged with sadness when I remember those hosts of opportunities, and how much better I might have used them. Richards was good enough to send me two papers on abdominal wounds, and a letter from which I gather he spends any spare time in learning Spanish. I doubt sometimes if our men, nobly as they have made sacrifices in many ways, realise what other sons of England were going through, a little more than a century ago. It was in 1810, I think, that *The Lady of the Lake* came out. A copy reached our army in the Peninsula. Five officers sat up all night taking turns to read it by a single candle almost blown out by the wind through the hut. When morning broke they had just finished it, notes and all. Only two got back to England. The grass at Pampeluna and Vittoria waves over two: a third lies in the churchyard at St. Etienne, which he had been told to hold to the last. Odgers may remember the story of Capt. Napier told to 'find the amputating place' at Badajos. If you remember Moffat, he also has the D.S.O., and mocks me by writing to me as 'My dear Master.' I well remember our first meeting. He joined late one November, and when I saw him copying an abstract of an anatomy lecture, he recalled the words of Bushby, fifty-seven years Headmaster of Winchester, of Bishop South, 'I see brains in that little boy, and my rod shall bring them out.' We did not meet again until he was H.S. to Sir H. Howse. That surgeon, if report was true, was not given to trusting and entrusting, and I recall

the pang it cost me to give Moffat an amputation of the thigh for a periosteal sarcoma. The girl, one of those rare instances of ladies met with even in the Boro', made a good recovery to die, a year later, of the frequent return in the lungs. Here in this out-of-the-way corner of the world one longs for any real information about the War. Some twelve years ago I interested myself in the boys of the parish. Out of sixteen, fifteen joined voluntarily. Three have been killed, one has been in the late pushes, carrying a bit of shell in his chest. Three are serjeants, one has the M.C. One, *spes gregis*, has, as I foretold from the first, a commission in the Queen's West Surreys, and has been wounded. From the Mont Dore Convalescent Home he sent me a list of books to choose from for him. A very striking instance of where in heaven or earth character really comes from. His father, somewhat bibulous, keeps the Black Dog public-house. His mother, ex-barmaid, has had syphilis (I was asked to see her for paraplegia which yielded to KI). His only sister, with J. Hutchinson's teeth, and S. osteitis, had a child before marriage. He, bright, intelligent and emphatically a gentleman in bearing, is now an Intelligence Officer, and humbles me to the dust.

"Odgers, who can, in a measure, realise my affection for much-loved Oxford, will be amused to hear that Time's whirligig has brought her associations back to the least worthy of her sons. On Sunday evenings I learn Latin hymns, mainly to replace the sugary, sentimental twaddle which is often dinned into one's ears, to tunes played without piety or poetry, at a rate as if all in church had to catch a train in ten minutes at a station two miles away. I lately came across a volume of translations by Chavasse. In my time at C.C.C. there were two 'Shavers, the Great and the Little Shaver.' The latter, with me at C.C.C., is now a blessed little Father in God at Liverpool, developing (as I am told) a decided tendency beneath his apron to what tailors euphemistically describe as 'the lower chest.' The elder, 'the big Shaver,' was Tutor at University and, as Proctor, made himself most unpopular, *circ.* 1866. Sons of Belial screwed him up with good old eight inchers one night. Proctorial duties wait neither for man nor mouse. No amount of perspiration plus cuss words availed with the screws (in two doors), and I remember the said Proctor descending into the finest street in the world, by means of a very long ladder, to the music of the jeers of a ribald crowd. However, he has found a better field in his translations of many of our best hymns, tho' it is a little odd to find Vergil, Horace, Ovid, Livy cropping up, as if to say, 'you never expected to



meet me again.' It may interest you to hear that I have lately, for the second time, declined to be Rector's Churchwarden. Our Rector is a man of pleasing and helpful personal example, but as narrow as the edge of my razors, without any of their acuteness. One only fit for a congregation of 'devout women not a few,' who prefers collecting money for altar-cloths instead of for a Lads' library; who never lets his flock near the Cross unless they come to Holy Communion, which in an agricultural parish will need a second Pentecost. I was asked a few years ago to join the Bench, and when I declined was urged to alter my mind. But Grattan said, 'Oaks are not transplanted at forty, and my father's son was not, when well over sixty, going to play at a profession of which he knew absolutely nothing. Thank you for your inquiries for my wife. She represented this house at a crowded wedding last week, and I hear that a lady said, 'Mrs. Jacobson was the sweetest-looking and the most nicely-dressed person in the church.' As for myself, I continue, as for thirteen years, to employ myself strenuously mind and body. Otherwise this dull stagnant place would be unbearable. But I have only to look back on a life of seventy-one years, singularly rich in blessings. With memories ever thankful and helpful, I am,

"Yours most truly,

"W. H. A. JACOBSON."

Jacobson was married, in 1891, to Miss Edith Mary Turgis of Ewelme, Oxfordshire, who survives him, and they had one son, Burton. The tragic loss of this only child, who was accidentally drowned in 1905 at the age of six, cast a shadow over Jacobson's later years and led to his immediate and complete retirement from the profession which he had adorned. In letters to his friends he gave the following reasons for giving up his work :

"The loss of this never-failing sunshine, morning and evening, a solace and encouragement in my very hard work, has led me to lay it down. . . . It will be some time before I have command of myself and as I have only two years more at Guy's, I am not going to take a slice out of it. And I am not only fifty-eight, but fifty-eight plus many years of habitual want of sleep. The work of the last year, going 120 miles to Guy's Hospital and back to Ewhurst four, often five and, in the taking-in week, six days a week, often changing wet things at the station, would have tried a younger and less jaded man. . . . Many will say I am unwise and so on. They do not realise the facts of the case; that is enough for me. The Examinership

is in a totally different category as to hard work, etc. But I have always held that if a man ceases to be in touch with his subject he should send in his resignation. It is for the Council to settle whether he should continue or not. If they say 'no' in my case I shall be a poor man, but I shall have peace and rest. At all events I have done what is straightforward and honourable. I am sure I am right about Guy's Hospital. I am an old tree and have had a staggering blow."

One who knew him in his retirement writes: "What impressed me most about Mr. Jacobson was his kindness to the poor of his parish of Ewhurst, the extent to which he denied himself the comforts—almost the necessities—of life in order that he might give weekly orders for meat, groceries, spirits, etc., to them. He was always ready, without any fee, to see any poor person with me, and to advise whether any further treatment was desirable. I think his retirement from professional work, after the death of his only son, was a mistake, as in many ways he was unsuited to country life—he did not hunt, shoot, fish or play lawn tennis, never having done these things in his youth. He devoted himself to his garden, which he made very beautiful, and from which he regularly sent fruit and vegetables to Guy's, but he often complained of the monotony of his life and would have liked to travel, but could not afford it because of his generosity. He realised far more clearly than most of us that war with Germany was inevitable, and that we were not prepared for it. This led him to give ambulance lectures in the surrounding villages: he declined any fee for these and would not even accept hospitality though he walked miles to give them. He was disappointed at not being asked to help at one or more of the War Hospitals, and I think that his talent ought not to have been wasted.

"His last illness came on insidiously, and many months before he died he told me that he was passing blood from the bladder at intervals and that he had passed a small fragment of a papilloma, but he considered himself 'too old for surgical intervention' and 'could not afford it.' I tried to persuade him to come and see you, but he would not be persuaded.

"To me he was always the kind friend who would take any amount of trouble to do me a good turn. When my father was ill he came to see him and was kindness itself, but when my father sent him a pedigree young dog Mr. Jacobson responded by sending him two dozen bottles of whisky. We sent him ferns from Wales for his garden, but he would not accept any more unless we allowed him to pay the railway charges."

The rest of his life Jacobson spent at Ewhurst, devoting himself to his wife, his garden and his poor neighbours. He was very good and kind to the children and had a great and lasting influence over them. On Sundays he taught them the Scriptures, with the aid of wonderful pictures, and immediately afterwards taught them how to shoot or play games. They adored him in return and made his latter days happier. Those who had the privilege of visiting him at Lordine Court can picture him still in his beloved garden, wonderfully but suitably clad, bending to his toil, proudly showing a rare and beautiful plant or bringing a weakling to a better place in the sunshine.

## AN ACCOUNT OF THE SHORT TUBE FOR THE TREATMENT OF STRICTURE OF THE ŒSOPHAGUS

By SIR CHARTERS SYMONDS, K.B.E., C.B., M.S., Consulting Surgeon to Guy's Hospital.

THE radiograms accompanying this note are, I believe, the first to be published showing the short elastic œsophageal tube *in situ*. Dr. Stebbing of the Lambeth Hospital has had tubes made infiltrated with barium sulphate, and has been good enough to furnish me with the illustrations (Fig. 1). Of the three radiograms, the first shows the situation of the obstruction an inch above the diaphragm, the second the tube incompletely inserted, having been pushed into position by a bougie, the third shows the funnel resting upon the stricture with the end in the stomach. The man from whom these radiograms were taken is still under treatment, is swallowing freely and is gaining ground. It is of importance to note the site of the stricture, as at one time the short tube was not thought to be suitable for the relief of obstruction at this level. The opaque tube, it will be seen, makes for greater accuracy in the management of tubage.

As the tubes have been in continuous use for thirty years, a brief account of the origin of the method of treating malignant disease of the œsophagus may be of interest, more particularly to readers of our Hospital Reports, since it was at Guy's that the method originated.

In the early eighties Krishaber of Paris introduced his long feeding tube; this was first employed by Mr. John Croft at St. Thomas's Hospital, and later by Mr. Arthur Durham at Guy's. The article in the second edition of Holmes's *System of Surgery*, published in 1870, on Diseases of the Larynx was written by Durham, who continued to take an interest in the subject. Cases of œsophageal obstruction came to the general surgical Out-patient department, and in 1883 and 1884 I used Krishaber's tube in several cases, but found that it caused a good deal of irritation and sometimes had to be removed after a few days. At the autopsy in one instance a deep ulcer was found at the back of the cricoid cartilage. The substitution of a silk web tube for the hard vulcanite variety gave some



(c) Same case with tube *in situ* after all of the opaque meal has entered stomach.



(b) Same case with tube *in situ*.



(a) Immediately after opaque meal.

relief, but did not wholly remove the trouble, for the stricture was in some cases so completely plugged by the tube as to prevent the swallowing of saliva, which caused considerable distress at night. It was to meet these objections that the short tube was suggested and arose in the following way.

In 1884 a case of malignant disease of the gullet came under my care in Job Ward. Krishaber's tube was introduced, but had to be removed in thirty-six hours on account of the distress occasioned. At that time I had as one of my dressers Campbell Gowan, who before taking to medicine had followed engineering, and was, moreover, a skilled mechanic. We discussed the possibility of making a short tube which would rest on the upper face of the stricture, and thus enable the patient not only to swallow, but also to enjoy the taste of his food. Five inches of a red-gum urethral catheter was attached to a box-wood cup

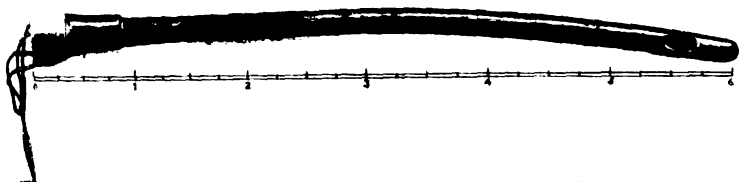


FIG. 2.

Short tube for oesophageal stricture: 1884 model.

by means of a ferrule of silver, and a length of silk passed through holes on each side of the cup completed the apparatus. On July 22, 1884, the tube was inserted by an ordinary bougie, and the man was immediately able to swallow, to our great joy and his own satisfaction. The relief from the constant expectoration was not the least of the benefits derived. After eight days the tube was removed, and was found in good condition; it was unfortunately thrown away. A second tube was made, this time with an ivory cup, and was inserted after an interval of twelve days, during which time the patient continued to swallow, for the stricture had become dilated by the first tube. This second tube is preserved, and as it possesses some historical interest has been added to our collection of antiquities (Fig. 2).

These two attempts having proved successful in permitting free deglutition, in removing the disadvantages of Krishaber's tube, and the silk not having caused any inconvenience, a more suitable form was made for me by Messrs. Down. It was in one piece of silk-web gum-elastic with a funnel-shaped upper

end. Since then the only change made in the tube is the provision of a second eye, which besides allowing a freer flow tends to prevent blocking of the lumen.

The next point was to construct a suitable introducer, and here Gowan's mechanical knowledge was of special value. It soon appeared that the pliable silk web was liable to bend in passing the pharynx, and that a rigid guide was required. Hence resulted the introducer still in use, viz. a whalebone stilette passing through a gum-elastic stem, furnished with an ivory bulb at the lower end, through which pressure is made on the funnel, and not by the whalebone stilette on the end of the tube, which might be perforated. In some cases a flexible bougie will pass a stricture while a rigid one will not; when this occurs the whalebone stilette is withdrawn for an inch or more, leaving a pliable extremity by which an awkward corner may be turned. The bulb is oval from above downwards and projects over the edge of the funnel, so as to facilitate withdrawal if an attempt at introduction fails. It should not be a complete oval, but have the sides flattened to prevent too intimate contact with the sides of the funnel. This feature was found valuable when open-end tubes were employed, as a twist released the bulb without dislodging the tube.

The patient was exhibited before the Clinical Society of London on January 23, 1885.\* A note of the case is published in vol. xviii. of the Transactions. Up to the time of his death on April 17, 1886, the tubes enabled him to swallow in comfort. A later note appended to this account is accompanied by an illustration showing the tube *in situ* and is taken from a specimen in our museum.

The department for Diseases of the Throat was established in 1886 and was placed in my hands. Cases of œsophageal obstruction naturally drifted to this quarter, so that one was able to bring before the Clinical Society on February 22, 1889 (vol. xxii.) two cases wearing tubes and to record an additional twenty-two cases, or twenty-four in all. The account says that no unpleasant consequences had attended the use of the tube, all had improved in health, and life had been prolonged from four to eleven months. In the management of these cases I received valuable assistance from my House Surgeon, Peter Paget, who acquired great dexterity in introducing the tubes.

As tubes are sometimes badly constructed it may be well to give a description of the correct pattern. It is made of silk-web gum-elastic in two lengths, of four and six inches. The lower

\* Six months after the commencement of treatment and wearing the silk web tube.

end is closed, while the upper is expanded into a funnel shape. The dimensions are catheter gauge Nos. 10, 12, 14, and 16. The wall remains the same thickness in all sizes, thus securing a larger lumen, and the inside of the tube should be polished to allow the easier flow of fluids. There are two eyes made as large as possible, half an inch apart, and beyond the further eye the tube should be hollow to the end. A length of plaited silk is passed through the perforations in the sides of the funnel across the lumen, and then the portion crossing the funnel is drawn out and knotted to the side pieces two inches above the funnel. The dimensions of the funnel should remain the same for all sizes, whether it be a No. 10 or 14. This is important, as the ivory bulb at the end of the introducer is constructed to fit the aperture.

The only alteration made in the tube since its introduction is the addition of the second eye.

A great merit of the elastic tube is that it can be introduced by anyone familiar with the use of the œsophageal bougie. So long as it remains free it may be worn without any inconvenience for several months. Should it become blocked, removal by means of the silk can easily be effected, the tube cleaned and reinserted, or replaced by one of larger size.

These tubes are most serviceable in disease affecting the middle of the gullet from a point ten inches from the teeth to fifteen inches, and as this is by far the most common situation it is applicable in the majority of cases. At a point nine inches from the teeth, in some cases there is just room enough for the funnel. In the lower end the tube is liable to be rejected by vomiting, but where this does not occur it has proved serviceable.

In a recent case under my care, where the disease affected this part and extended along the cardia of the stomach, a tube was worn with intervals over a period of eighteen months, and, as mentioned above, the patient from whom the radiograms were taken is another example.

When extension of disease takes place into the trachea, the lung or mediastinum, fluids passing by the side of the tube naturally cause irritation and a short tube is of no further use. Then the long silk web or a rubber tube \* becomes necessary.

The danger of perforation of the growth by a bougie is very slight, provided two cautions be observed: first, never to use any force, and second, to withhold pressure when the patient strains against the bougie.

The moment the patient takes a deep breath the bougie may be advanced with safety, and this moment must be waited

\* First employed by Mr. James Berry (*St. Bart. Hosp. Rep.*).



for. Intolerance will occasionally prevent the insertion of a tube on the first attempt, but this is soon overcome, and I have never been obliged to abandon the method on this account.

With one or other form of tube I have found it possible to render these cases more comfortable and content than by a gastrostomy. The object of this note does not permit an account of the treatment of the various forms of malignant disease of the œsophagus, nor of the resulting complications. That the method of tubage is in general use is a sufficient tribute to its value, and my own experience justifies the statement that, provided advice is sought in the early stages gastrostomy should never be required.

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# NERVOUS SYMPTOMS DUE TO ALKALÆMIA AND RENAL INSUFFICIENCY, FOLLOWING OBSTRUCTION IN THE REGION OF THE PYLORUS, AND THE ALKALINE TREAT- MENT OF DUODENAL ULCER

## INTRODUCTION

It has long been known that pyloric obstruction is an occasional cause of severe nervous symptoms. The most familiar variety is tetany, which was first recorded as a sequel of pyloric obstruction by Kussmaul in 1869, but headache and other minor symptoms, which may culminate in coma—the “coma dyspepticum” of Von Sakset—and epileptiform convulsions, may also occur and give rise to a clinical picture closely resembling uræmia. The “uræmic” group of symptoms has been described from time to time during the last twenty-five years, but it does not appear ever to have been as widely recognised as the more striking but no more common tetany.

Many theories have been put forward to explain the occurrence of tetany and the other nervous symptoms in pyloric obstruction. They fall into three groups, the symptoms being supposed to be caused (1) by the absorption of toxins produced by stagnating food in the dilated stomach, (2) by dehydration caused by excessive loss of water in vomiting, and (3) by a reflex from the distended stomach. All these theories are now known to be devoid of foundation. Although the condition has not yet been completely explained, it is recognised that there are two principal factors: (1) alkalæmia, shown by low chloride and high bicarbonate in the blood, due to loss of hydrochloric acid in the vomit, and (2) renal insufficiency, as shown by the excess of urea and creatinine present in the blood, and impaired phenol-sulpho-naphthalein excretion (Brown, Eusterman, Hartman and Rowntree);<sup>1</sup> a degenerative form of nephritis was found post-mortem in six fatal cases described by Brown and his colleagues.

The introduction of the Sippy method of treating gastric and duodenal ulcer with sufficiently large doses of alkalis to produce complete, or almost complete, neutralisation of the free acid in the stomach throughout the day has been followed on rare occasions by severe nervous symptoms which have even proved fatal. Sippy himself never referred in any of his publications on the subject to this toxæmia, although the cases of partial

pyloric obstruction in which he claimed to get excellent results are those in which this complication is said by Hardt and Rivers<sup>2</sup> to be most likely to occur. In the cases observed at the Mayo Clinic by Hardt and Rivers the patient was at first unduly introspective and nervous. He then complained of difficulty in taking his milk. After a time headaches, nausea and vomiting occurred. The patient became apathetic and drowsy, and in two cases death ensued. In every instance the urea in the blood became greatly increased, and in several cases albumen and casts appeared in the urine. In some there was evidence of kidney disease before the symptoms developed, and in the two fatal cases described by Hardt and Rivers an acute exacerbation of a chronic nephritis was found after death. These toxic symptoms may develop in patients with partial pyloric obstruction, who have in consequence continuous hypersecretion, considerable quantities of acid being often lost by vomiting; corresponding with this, Hardt and Rivers found a rise in the plasma bicarbonate. Thus this group of cases has many similarities to that in which nervous symptoms follow pyloric obstruction apart from any special line of treatment, the chief factors being alkalæmia and renal insufficiency.

In a recent paper on alkalæmia Ellis<sup>3</sup> discusses the biochemical factors involved in these various conditions, and refers to Blum<sup>4</sup> as having reported the production of epileptiform convulsions from the intravenous injection of sodium bicarbonate in patients with diabetes coma in 1913, but as long ago as 1905 Beddard drew attention at Guy's Hospital to the danger of poisoning in the alkaline treatment of this condition. Howland and Marriott, Harrop and Healy have all reported cases of tetany following treatment of various conditions other than gastric or duodenal ulcer with large doses of sodium bicarbonate. In several of their cases and in Ellis's first case deficient renal excretion also appeared to play an important part in the production of the toxic symptoms.

In the following pages a group of three cases is recorded by Mr. L. Wynn Houghton, in which uræmic symptoms resulted from obstruction in the neighbourhood of the pylorus in the absence of any alkaline treatment, and a further series of seven cases is recorded by Dr. J. F. Venables, in six of which obstruction was absent, but in which a modified form of the alkaline treatment had been given and was probably the exciting cause of the symptoms. Finally, two cases of pyloric obstruction with chemical evidence of alkalæmia and renal insufficiency but without nervous symptoms are described by Mr. N. L. Lloyd.

A. F. H.

## A. THREE CASES OF TOXÆMIA FOLLOWING OBSTRUCTION NEAR THE PYLORUS

By L. WYNN HOUGHTON, Assistant House Surgeon, Guy's Hospital.

*Case 1. Tetany and uræmic symptoms caused by obstruction of the jejunum after a gastro-enterostomy ; operation ; recovery.*—A. L., male, aged 44, was operated on in Guy's Hospital in 1918 for a perforated gastric ulcer, posterior gastro-jejunostomy being performed at the same time. He made a good recovery and was quite well until May 1924, when he suddenly began to vomit after meals. The vomiting increased in frequency and amount ; he noticed that he often brought up more than he had taken at the previous meal and that the vomit was usually bile-stained.

When seen in the Out-patient Department on July 19th, 1924, well-marked gastric peristalsis was visible in the epigastrium, but there was no pain.

He had definite signs of tetany of carpo-pedal spasm type, and was in a drowsy condition and dull mentally, but he was not then so severely ill as to be unfit to go home and wait for four days for a vacant bed.

He was admitted under Dr. French on July 23rd, a good deal worse than he had been on the 19th. He was very thin and wasted, and his face was pale with a tinge of cyanosis. He was semi-comatose and answered questions slowly and with great difficulty, often correcting himself.

His respiratory and cardio-vascular systems seemed normal ; the blood pressure was 110 mm. Hg. systolic, 90 mm. Hg. diastolic. His mouth was dry, his tongue brown and furred, and his breath very foul-smelling. There was a linear scar on the abdomen above the umbilicus resulting from the operation performed for perforated ulcer in 1918, and a smaller one above the pubes, where a drainage tube had been used. There was a well-marked gastric succussion splash over a wide area, with visible gastric peristalsis but no vomiting.

Chvostek's and Trousseau's signs were negative, but there was muscular irritability, and when he tried to grasp anything with his hand he showed carpal spasm.

The urine had specific gravity 1020 and was slightly acid. Only a faint trace of albumen was present and no casts were found microscopically. The albumen seemed to be of little importance at this time, and a diagnosis of gastrectasis from pyloric stenosis due to a healed ulcer, with tetany as a secondary phenomenon was made.

Four hours after admission the stomach was washed out with warm saline solution, and a huge quantity of bile-stained, sour-smelling material obtained. The stomach lavage was repeated the following morning, but there was no alteration in the patient's condition. On July 25th he was worse with deep coma, hissing respiration and cyanosis. The urine showed a decided trace of albumen without casts, but the blood-urea was found to be 0.45 per cent., or about fifteen times the normal, so

that the slight albuminuria forthwith assumed a much greater degree of importance, and it was difficult to escape the conclusion that uræmia was responsible for a great part of the mental and nervous symptoms.

Lumbar puncture was performed at once, and about 12 c.cm. of cerebro-spinal fluid drawn off. It was under slightly increased pressure, had the same percentage of urea as the blood, and the protein was 0.06 per cent. The patient was then given pulv. jalapæ co. gr. xxx, an axillary injection of saline solution, and a hot-air bath.

The next morning his condition was about the same, so 300 c.cm. of blood were removed, and 600 c.cm. of saline solution given intravenously. The axillary saline injections and hot-air baths were continued, and on the 28th he was partially conscious, taking fluids by the mouth and passing plenty of urine, but the blood-urea was still 0.40 per cent.

The following day the visible peristalsis returned and he complained of abdominal pain. As he was able to take fluids without vomiting he was given an opaque meal and screened. The stomach was very large, with powerful and deliberate peristalsis, but some barium was seen passing through the pylorus. There was no sign of a gastro-jejunostomy opening.

On August 3rd he was sitting up in bed reading a book, was taking solid food and not vomiting. The blood-urea was 0.05 per cent.

He took good quantities of food for a fortnight, and on August 16th his stomach was washed out, and Mr. R. Davies Colley opened his abdomen under open ether anæsthesia. The stomach was greatly dilated; there were a few adhesions round the pylorus, and it was discovered that a posterior gastro-jejunostomy had been performed at the operation for perforated ulcer in 1918, but that the jejunum had become kinked at the stoma so that no food could leave the stomach by that route. There was hardly any pyloric stenosis and the duodenum was greatly dilated. A duodeno-jejunostomy was performed.

The patient recovered well from the operation, and when discharged on September 3rd was felling well, was free from any mental, tetanoid or nervous symptoms, was putting on weight and taking full diet. The urine was free from albumen and the blood-urea figure normal.

*Case 2. Uræmic symptoms with suppression of urine following malignant pyloric obstruction.*—Lucy H., a married woman aged 42, was admitted under Mr. Philip Turner on November 9th, 1924, as a case of pyloric stenosis.

She had had her right breast removed seven years ago, but her husband could not say for what this was done. Otherwise she had always been healthy until four months before admission, when she started having epigastric pain of an irregular nature. This continued for two months, and she then began to vomit occasionally after meals. The vomiting became more frequent, and for the last week before admission she vomited everything

she took, whether solid or fluid. She had lost a great deal of weight in the last two months.

On admission she was very thin, her cheeks had a bright pink flush, and she was wandering mentally, although there was nothing approaching coma. Her pulse was 80, regular, but very weak at the wrist, the temperature 97° F., and respirations 20.

The abdomen was flaccid; there was no distension, no tenderness, and nothing abnormal could be palpated. The stomach seemed to be dilated. The bowels were constipated, and the patient was unable to pass any urine.

The central nervous system was normal, and none of the signs of tetany could be elicited.

An enema was given shortly after admission but was not retained. Whatever she took by mouth she vomited at once. A catheter was passed and about two drachms of urine obtained. It contained a trace of albumen, but no sugar or blood. Her blood-urea was found to be 0.17 per cent., or about six times the normal.

The patient was restless during the night and hardly slept at all, but seemed a little better the following morning and was able to keep down some water. Her mental condition remained the same.

Two pints of saline solution were given subcutaneously.

Twice during the day a catheter was passed, but no urine could be obtained. A hot-air bath was tried for 1½ hours, but failed to produce diaphoresis.

The patient passed a restless night, and at 7 a.m. the following morning she became unconscious, cyanosed, and had two violent fits at an interval of ten minutes. She had the typical hissing respiration of uræmia.

Lumbar puncture was performed at 7.30 a.m. and a test tube full of cerebro-spinal fluid taken. It was under slightly increased pressure and contained slight excess of albumen. Her pulse at this time was imperceptible at the wrist, and the heart sounds were very feeble. She was quiet for an hour and a quarter, but had another fit at 8.45 a.m. and died almost immediately.

At the post-mortem a diffuse growth involving the whole pyloric end of the stomach was found. The mesenteric glands were enlarged, but there were no secondary deposits in the liver. The kidneys appeared normal macroscopically, and microscopically nothing more than cloudy swelling of the tubular epithelium was found.

*Case 8. Fatal uræmia following incomplete pyloric obstruction caused by a chronic gastric ulcer.*—Mrs. W., aged 50, was first seen on December 23rd, 1923. She had been vomiting more or less continuously for the last fourteen days, and latterly had been unable to keep down even milk and water. A year previously she had had a similar though not such a prolonged attack, and her doctor thought the condition was mainly a neurosis. In the interval she had not been fit, had not eaten much, and had lost some weight. With the vomiting there was

only very little pain, which was situated around the umbilicus. The patient was rather emotional and wept on being questioned.

Her tongue was moist and slightly furred. Her pulse was 108 and very thready, but the systolic blood pressure was 140 mm. Hg.

The abdomen was quite flaccid and not distended; nothing abnormal was felt, and there was no tenderness.

Examination of the lungs, heart, and central nervous system showed nothing abnormal, but the tendon reflexes were exaggerated. There was no albuminuria.

A diagnosis of functional vomiting was made, but on account of the thready pulse and prolonged shortage of fluids she was admitted into the hospital.

The vomiting continued until she had gastric lavage, after which it stopped. The x-rays showed only considerable ptosis of the stomach, but there was gastric stasis up to eight hours.

She was given an abdominal belt, and after being reassured she was discharged.

She was readmitted in February 1924 with a return of symptoms. Mental unbalance had developed. Great dilatation of the stomach was now present, four pints being removed by a tube. The urine contained albumen and casts. The blood urea was  $2\frac{1}{2}$  times the normal.

Although the gastric symptoms diminished, the patient became gradually weaker and died.

At autopsy a chronic ulcer was found on the gastric side of, but not occluding, the pylorus. The stomach was not dilated, but the mucous membrane was inflamed. The kidneys appeared normal to the naked eye.

I am indebted to Dr. French, Mr. Turner, and Dr. Ryle respectively for permission to publish these three cases.

## B. SEVEN CASES OF ALKALOSIS FOLLOWING ALKALINE TREATMENT OF DUODENAL ULCER

By J. F. VENABLES, M.D., Assistant Physician, New Lodge Clinic.

*Case 1. Duodenal Ulcer ; Pyloric Stenosis.*—M., 50. The patient had a long history of duodenal ulcer, and during the last few months the most marked symptom had been excessive vomiting. The vomit often contained food that had been taken many hours previously. A fractional test-meal showed well-marked delay in the stomach. The volume of the resting juice was 180 c.c. An x-ray examination confirmed the presence of a mild degree of gastric delay.

Medical treatment was tried in the hope that the pyloric obstruction might be due to inflammatory oedema and reflex interference with the pyloric sphincter, as we have had several cases in which complete recovery has followed medical treatment of pyloric obstruction due to an active ulcer, and in

three instances with visible peristalsis. The patient's condition at first improved. Pain disappeared and the vomiting ceased. After about ten days, however, he became very depressed, complained of severe headache, vomiting recurred, and complete anorexia was present. The patient also complained of intolerable itching of the skin and was extremely irritable. His condition did not improve when alkalies were excluded. The blood urea figure gradually rose from 0.9 grms. per 1000 to 3.4 grms. per 1000 c.c. The patient became comatose, and died in July 1922 without having shown any response to treatment. This was the first case we had observed of nervous symptoms developing during the alkaline treatment of an ulcer, and the uræmic symptoms were not immediately recognized as being caused by alkalosis.

*Case 2. Duodenal Ulcer.*—M., 45. The patient had complained of right-sided pain for eight years. Two previous operations had been performed—appendicectomy and fixation of the cæcum. Neither gave relief, although occasionally there would be a period of freedom from symptoms for as long as six months.

The symptoms were not typical of duodenal ulcer, but the x-rays showed a definitely deformed duodenal bulb, and occult blood was constantly present in the stools. The stomach emptied within the normal time limits, as shown with a test-meal and with an x-ray examination. The systolic blood pressure was 115 mm. of mercury.

The patient was put on the full ulcer treatment, but did not improve, doubtless owing to the fact that the ulcer was adherent to the head of the pancreas; this fact was demonstrated at the subsequent operation.

After fourteen days' treatment the patient's general condition deteriorated. He became drowsy; his skin and tongue became very dry; he complained of headache, anorexia and nausea.

The blood urea was found to be 1.06 grms. per 1000 c.c., or three times the normal. The administration of alkalies was at once stopped, and the patient's condition showed an almost immediate improvement. One week later the blood urea figure had fallen to 0.43 grms. per 1000 c.c. and the toxic symptoms had disappeared. The urine throughout was normal, and there was nothing to suggest any preceding renal inefficiency.

*Case 3. Jejunal Ulcer.*—M., 55. The patient had had symptoms of duodenal ulcer since 1909. Nephrectomy was performed for cystic kidney in 1912. This was followed by numerous attacks of bacilluria. A gastro-enterostomy was performed in 1916.

The patient had a severe hæmatemesis in 1919, apparently from a jejunal ulcer, and was treated for three months with satisfactory results.



In 1922 he had a recurrence of symptoms, and on investigation it was found that he had a jejunal ulcer. There was constant occult blood in the stools; the tenderness was found on x-ray examination to be confined to the site of the anastomosis, and a test-meal showed a high normal curve for free acid.

The patient was put on full ulcer treatment on November 15. Toxic symptoms consisting of headache, drowsiness and anorexia appeared within a week, and a blood urea estimation on November 22 gave a figure of 1.02 grms. per 1000 c.c., or fully three times the normal. The blood urea one week later, after alkalies had been excluded, was still 1.1 grms. per 1000 c.c. On December 13 a further estimation gave a figure of 0.59 grms. per 1000 c.c.; the toxic symptoms had by this time disappeared.

No casts were detected in the urine, but a slight cloud of albumen was constantly present, associated with a small amount of pus. No previous estimate of renal efficiency had been made, but probably 0.59 grms. per 1000 c.c. was the average figure for the blood urea, and there appears therefore to have been some degree of inefficiency before alkali treatment was commenced.

*Case 4. Duodenal Ulcer.*—M., 62. The patient had a very long history of duodenal ulcer with repeated hæmatemesis. The diagnosis was confirmed by the x-ray examination and test-meal, and by the presence of occult blood in the stools. Neither the x-ray examination, which revealed a very deformed duodenal bulb, nor the test-meal, which showed marked hyperchlorhydria, suggested the presence of any gastric delay. The systolic blood pressure was 112 mm. of mercury.

Full ulcer treatment was commenced on July 12. A previous blood urea estimation on July 7 gave a slightly raised figure, 0.46 grms. per 1000 c.c. By July 20, one week after treatment had commenced, the patient complained of nausea, headache and complete anorexia. The alkalies were stopped and a further blood urea estimation was made. The figure was 0.71 grms. per 1000 c.c., a definitely raised figure as compared with the estimate of eight days before. The symptoms quickly subsided when alkalies were withheld, and on August 13 the blood urea was 0.53 grms. per 1000 c.c.

The urine was at all times normal, but in view of the slightly raised blood-urea figure before treatment was commenced and again after all alkalies had been withheld for over a fortnight, it is probable that slight renal inefficiency was present.

*Case 5. Duodenal Ulcer.*—F., 42. The patient had a history of some years' duration suggesting duodenal ulcer, and had had one severe hæmatemesis. The diagnosis was confirmed by the discovery with the x-rays of a deformed bulb. Neither the x-ray examination nor the test-meal, which showed marked hyperchlorhydria, gave any evidence of gastric delay. The systolic blood pressure was 145 mm. Hg.

Full ulcer treatment was started on February 4. On February 16 the patient complained of feeling intensely depressed; in fact she was crying without cause, a most unusual occurrence, as she was an extremely cheerful and plucky patient. At the same time she complained of nausea, anorexia and a mild headache. A blood urea estimation at this time gave a figure of 0.49 grms. per 1000 c.c., definitely above the normal reading of 0.3 grm. Alkalies were at once excluded, and all symptoms disappeared within a few days. This patient was subsequently able to tolerate small doses of alkali, and she completed her treatment without any repetition of the unpleasant symptoms:

*Case 6. Duodenal Ulcer.*—F., 49. The patient had a history of long-standing indigestion. A duodenal deformity was demonstrated on x-ray examination. Occult blood was constantly present in the stools, and on one occasion there had been melæna. Neither the x-ray examination nor the test-meal, which gave a normal curve, showed any evidence of gastric delay. The systolic blood pressure was 120 mm. Hg.

Toxic symptoms developed one week after treatment with full doses of alkali had been started. The symptoms were mild, consisting of depression, headache, nausea and anorexia. A blood urea estimation at this date gave a figure of 0.45 grms. per 1000 c.c. One week later after all alkalies had been excluded the blood urea figure was 0.33 grms. per 1000 c.c., and all symptoms had disappeared. This patient thus developed symptoms with only a slight rise of blood urea.

*Case 7. Duodenal Ulcer, Chronic Appendicitis and Gall-Stones.*—M., 61. The symptoms of indigestion had been present for many years. An x-ray examination revealed a deformity of the duodenal bulb; the stomach emptied itself within normal time limits. The normal emptying rate was confirmed by a fractional test-meal, which gave a normal curve for free acid. The systolic blood pressure was 140 mm. Hg.

Full ulcer treatment was started on November 17. Almost a fortnight later the patient complained of headache and anorexia, and his skin, which was very dry, itched considerably. The blood urea on December 3 was 0.84 grms. per 1000 c.c., compared with 0.50 grms. per 1000 c.c. on November 12, before any treatment had been instituted. Alkalies were withheld, and the symptoms quickly disappeared. In this case there was some evidence of slight renal inefficiency before the treatment was started.

#### SUMMARY AND CONCLUSIONS

1. Toxic symptoms occasionally develop in the course of treatment of duodenal ulcer with large doses of alkalies. This

has never occurred in our experience in cases of gastric ulcer. Curiously enough, toxic symptoms have never occurred in any of Dr. Hurst's hospital patients treated in exactly the same manner, although the number under observation has been approximately the same.

2. The symptoms generally appear within seven, and always within fourteen, days of commencing the alkaline treatment. Those common to all the cases were anorexia and depression; both developed early. Other symptoms were headache, irritability, pruritus and dryness of the skin and tongue. Only in the fatal case did more severe symptoms develop; the patient presented the picture of severe uræmia with unconsciousness for eight hours before death.

3. One of the seven cases had partial pyloric obstruction; this was the only patient with a history of prolonged and severe vomiting. In all the others the x-rays and fractional test-meal showed a normal rate of evacuation of the stomach. This is in contrast with the findings of Hardt and Rivers.

4. No case has occurred in a patient under 40 years of age, the average age of this series being 52.

5. In every case the blood urea was found to be abnormally high when estimated after the development of symptoms.

6. Only one patient had a definite history of renal disease. Two others had a slight excess of blood urea before treatment was begun, the figure rising still higher at the end of a week's treatment. In the others no previous estimation of urea had been made, but in two the fall after the treatment had been discontinued was incomplete, so that perhaps some renal insufficiency had previously been present. The urine was only abnormal in the patient with old standing cystitis and pyelitis. The highest blood-pressure recorded was only 140 mm. of Hg. Three of the patients had rather a low blood-pressure for their age.

7. Gastric analysis did not show any constant type of curve, though the tendency was towards a high figure. Three patients had well-marked hyperchlorhydria, two gave high normal curves, and the two others had average normal curves.

8. The following practical conclusions can be drawn : (i) In the presence of even a mild degree of renal disease, a duodenal ulcer should be treated by diet, olive oil and atropine, without alkalies. (ii) In patients over forty with no signs of renal disease the blood urea should be estimated before beginning treatment, and if it is above normal alkalies should be avoided or given only in comparatively small doses. (iii) If any suspicious symptoms arise in a patient under treatment for duodenal

ulcer, the alkalies should be omitted until the blood urea has been estimated. If it is raised, no more alkalies should be given. (iv) When an ulcer is complicated by even slight pyloric obstruction, the alkaline treatment should only be given if the blood-urea is normal; if it is normal, the slightest sign of nervous symptoms should be regarded as a warning to discontinue the use of alkalies until the blood-urea has again been estimated; if it is now raised and the ulcer does not show signs of healing under simple dietetic treatment without alkalies, a gastro-enterostomy should be performed without further delay after preliminary gastric lavage and administration of large quantities of saline solution by rectum or subcutaneously.

### C. TWO CASES OF PYLORIC OBSTRUCTION WITH ALKALÆMIA AND RENAL INSUFFICIENCY BUT NO NERVOUS SYMPTOMS

By N. L. LLOYD, Medical Registrar, Guy's Hospital.

*Case 1. Pyloric Stenosis, due to Ulcer, with Alkalæmia.*—W. M., aged 46 years, was admitted under the care of Dr. Hurst with a nine months' history of vomiting at irregular intervals, bringing up large quantities at a time, the matter often consisting of food which he had taken the day before.

Clinical examination, confirmed with the x-rays and fractional test-meal, showed that he had gastric delay with pyloric stenosis.

The gastric acidity was within normal limits, the maximum total acid being 70 and the free acid rising to 37.

The urine showed no abnormality, and was acid in reaction.

Shortly after admission the blood urea was 0.06 grm. per 100 c.c. and the plasma bicarbonate 0.0446 Molar  $\text{NaHCO}_3$ , or one-third above normal (0.032–0.035) (A. Osman).

Treatment was given in preparation for operation, the patient's stomach being washed out daily, and he was given glucose solution by rectum. One week later his blood urea had fallen to 0.017 grm. per 100 c.c.

An operation was performed three days later, and multiple duodenal ulcers with pyloric stenosis were found. Mr. R. P. Rowlands performed a gastro-jejunostomy.

Five days after the operation the plasma bicarbonate was found to be reduced to 0.0325 Molar  $\text{NaHCO}_3$ , or within normal limits.

In this case there was ample acid in the gastric secretion to account for the development of alkalosis on vomiting. It is clear in this case, as in Wynn Houghton's first case, that the

disturbance of renal function was temporary and recoverable, being due apparently to the alkalosis and not renal disease.

*Case 2. Malignant Pyloric Stenosis with Alkalæmia.*—T. P., aged 55 years, was admitted with a history suggestive of pyloric stenosis associated with much vomiting, and clinically and at the subsequent operation this was confirmed, the stenosis being due to a large growth.

Examination of the food vomited showed no free acid and a low total acidity. A fractional test-meal was not performed. His urine showed no abnormality and was acid in reaction.

He was prepared for operation by having his stomach washed out, and he was given glucose by rectum.

A few days after this treatment had been begun, and at a time when there had been no vomiting for a day, the blood-urea was found to be 0.037 grms. per 100 c.c., and the plasma bicarbonate 0.039 Molar  $\text{NaHCO}_3$ , both being raised above the normal limits (A. Osman).

Laparotomy revealed an inoperable growth. The patient died a few days later. The blood examinations were not repeated.

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## THE ÆTIOLOGY AND CLASSIFICATION OF THE COMMON NEUROSES

By C. P. SYMONDS, M.D., Assistant Physician for Nervous Diseases,  
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Of the subject of this paper it might be said that none is of greater interest to the medical practitioner, none more obscured by speculative theory and confusion of words. The neurotic is a weekly—if not daily—event in the practice of physician and surgeon, specialist and general practitioner. His symptoms, despite their superficial variety, are often so well defined that they are spoken of as “characteristic” and “typical.” Yet discussion between medical men, of ætiology and treatment, is too often lost in the quicksands of shifting terminology, or swept into space by Freudian gusts. Nor can the medical student take up half a dozen manuals of the subject without finding as many different meanings attached to such terms as “neurasthenia” and “psychasthenia.”

It has seemed to the writer worth while to record this outline of the ætiology and classification of the psychoneuroses for two reasons. First, that it is based upon physiology and common-sense; second, that it avoids the use of certain terms which from constant wear have lost whatever face value they originally possessed.

The following notes, though they are the product of personal thought and experience, fall partly into the mould given by Ross in his *Common Neuroses*, to which the writer is much indebted.

### ÆTIOLOGY

A neurosis, or, as the public have it, a “nervous breakdown,” is the expression through the nervous system of a failure on the part of the organism as a whole to adapt itself to environmental stress.

In every case four possible factors have to be considered :—

1. Heredity.
  2. Faulty habits of mind.
  3. Physical disease.
  4. Emotional stress.
- Factors 1 and 3—Heredity and Physical disease, call for no further discussion in this paper. We are all aware of the importance of the family history in estimating the ability of a patient to overcome the ordinary—or extra-

ordinary—difficulties of life, and we have most of us had personal experience of the effect of physical illness upon patience and judgment. The other causal factors, faulty habits of mind—in the main ignorance and self-deception—and emotional stress, need to be studied in greater detail. Psychological theory is in its infancy: much of it is still in the speculative stage. There are, however, certain phenomena whose existence is little disputed, and upon which a useful conception of psychopathology may be based. These are :—

### 1. *The Emotional Reaction*

Whenever the emotions are strongly aroused by any external or internal stimulus there results a state of mental excitement, which has an unpleasant quality and is termed *anxiety*. This normally persists until the situation has been met, and is then followed by a sense of relief.

In proportion to the amount of anxiety, reflex bodily changes occur. These are effected chiefly through the autonomic nervous system and are beyond voluntary control. They may be conveniently classified as follows :—

*Circulatory System.*—A sudden and severe emotional stimulus may cause temporary cessation of the heart beat through vagal inhibition. This results in loss of consciousness, and is responsible for the common faint or swoon of emotional origin.

Less severe stimulation causes acceleration of the pulse with forcible thumping action of the heart, and throbbing of the large vessels.

The peripheral arterioles and capillaries under the influence of severe emotion may contract with resultant pallor, but more commonly they dilate, the phenomenon of blushing being due to this cause.

Except in the case of severe emotion with vagal inhibition the blood pressure is raised.

*Alimentary System.*—The secretion of saliva is diminished. There may be spasm of the pharynx and œsophagus. The movements and tone of the stomach and intestines are diminished, together with spasm of the pyloric and ileo-colic sphincters. In acute emotion vomiting may occur. Gastric secretion is abolished or reduced. The pelvic colon contracts and the anal sphincter is relaxed, leading sometimes to evacuation of the bowels.

*Respiratory System.*—The respiratory movements are increased in depth and frequency, and with this there may be a sensation of “suffocation.”

*Genito-Urinary System.*—The secretion of urine is increased

and the bladder contracts. This leads to a call to micturition, or in extreme cases there may be involuntary relaxation of the sphincter with incontinence of urine. In males prostatic secretion and emission may occur.

*Endocrine Glands and Metabolism.*—The output of adrenalin is increased, and this in turn may cause further stimulation of the organs innervated by the sympathetic system. The blood sugar is raised and glycosuria may occur. Metabolism generally is increased.

*Nervous System.*—Besides the mental condition of anxiety which fills the mind to the exclusion of purposeful thought and of sleep, there may be tremor of the limbs and sensations of numbness. Discomfort or pains in the head are commonly experienced, and are probably due to changes in the cerebral circulation. The pupils are dilated, and the eyes may be protruded.

*Skin.*—The erectors of the hair follicles may be excited, producing the phenomenon commonly described as “goose flesh.” Profuse sweating may occur.

The emotional reaction, if it persists for any length of time, is accompanied by an unusual sense of mental and physical fatigue.

## 2. The Conditioned Reflex

Pavlov has shown in animals that by means of experience a previously indifferent stimulus may become so closely associated with an effective one, as itself to become effective. The experiment in which the sound of a bell was in this way made effective as a stimulus to the salivary reflex is familiar to students of physiology. In the same way an indifferent stimulus may through association become capable of producing the emotional reaction.

The association may be produced either by frequent repetition or by the coincidence of particular circumstances with an especially severe emotional reaction. Many examples might be quoted of such an association leading to a conditioned emotional reaction. During the recent war men who had been exposed for long periods to occasional shell-fire would afterwards exhibit tremor and palpitation at any loud and unexpected noises. A woman who was summoned to the telephone to hear that her husband had been killed suffered for several months afterwards from a sensation of extreme anxiety and palpitation whenever she was called to the instrument.

Similar emotional disturbances may be experienced by a man who has met with an accident while driving a motor-car when next he takes the wheel, even though he is an experienced driver



and the road is clear. Such associations are usually lost with further experience in the same way as the simpler conditioned reflexes established in animals can be broken down by further training.

### 3. *The Unconscious Mind*

To anyone who has reflected upon the matter it must be evident that one is not fully aware at a given moment of all that is going on in the mind, and further that one may at times act in a manner which implies purposeful thought without being aware of any such thought or purpose. Thus, for instance, a man absorbed in his work may remain seated at his desk for some time after the gong has summoned him to dinner. Given that his auditory apparatus was intact, an observer might reasonably conclude that the man had decided to postpone going in to dinner in order to complete his task. The subject, however, might honestly declare that the reason of his delay was that he had not heard the gong. Or, again, a man immersed in deep thought may put a letter in a post-box, yet remain completely unaware that he has done so. In these instances the rejection of the summons to dinner and the posting of the letter provide examples of unconscious mental processes.

Such phenomena become more intelligible when the nature of attention is considered. Attention may for the moment be compared to a searchlight whose beam is focussed upon a stage on which are represented the mental processes. No part of the stage is ever empty of incident, but its total area is large in comparison with the circle of illumination. Events in the centre of the beam are brightly lit, those in the periphery more dim, while the greater part of the drama is enshrouded in darkness.

The illuminated area may be taken to be the field of consciousness. The remainder of the stage represents the unconscious mind. Returning to the instance in which the dinner gong was ignored we may say that this event took place upon an unlit part of the stage. It did not come within the circle of illumination or field of consciousness, but remained an unconscious mental process.

We may now carry the analogy a step further and imagine the searchlight to be mounted upon a pivot so that it may easily swing in this or that direction. Let us assume also that objects and episodes upon the stage have power to swing the searchlight towards them or away according to the emotions with which they are invested. Events which are associated with the emotions of curiosity or desire tend to attract the beam (positive influence); those which inspire shame or fear repel it (negative influence).

Some objects may have so powerful an attraction that they continuously occupy a central place in the field: others may exert so strong a repelling force as never to come within the area of central illumination, since whenever they appear upon its margin they cause the searchlight to swing away from them.

If at a given moment the centre of the beam be upon episode or object A, with a low positive influence, and upon the margin of the field appears object B, with a more powerful attraction, the tendency of the light will be to swing towards B, unless the attraction of A is in some way increased.

For example, we may take the case of a schoolboy on a summer afternoon bent upon his preparation work with the object of completing it as soon as possible. For the moment his book occupies the centre of the field of consciousness. Then as it were faintly the sounds of cricket outside appear upon the margin of the field. The beam of attention swings automatically in the direction of cricket to the exclusion of the work. Then comes the thought that the quicker his task is done the sooner he will get to his play. So the attractive value of the work will be enhanced and the beam of attention again is focussed upon it.

The emotions themselves form a part of the mental activities that proceed upon the stage, and lie to a great extent outside the field of consciousness. Thus a man may be unaware of the motives which direct his attention. This is often apparent in scientific work. The aim of the scientific worker is to discover the truth. He is, however, only too often influenced unwittingly by the desire to prove his own theory correct. Such facts as are consistent with that theory attract attention. Those which are contrary to it escape his notice. The selfish motive which is responsible for this is itself repellent and remains unseen.

This tendency for a man to remain unconscious of his own emotions if they are of a repellent nature is of great importance in the causation of the psychoneuroses. For instance, in the recent war a man whose training had taught him to regard the emotion of fear as tantamount to cowardice would sometimes remain oblivious to the presence of this emotion within himself. The emotional reaction would then be present without cognisance of the emotion, and the individual would be driven to explain the phenomena of palpitation, sweating and so on as evidence of bodily disease.

Furthermore, he might be led by his fear to evade his duty without being conscious of the motive which impelled him. Physical disease under such circumstances offered a way of escape, and in this state of mind a man would, in the face of

all reasonable considerations, accept the least suggestion of bodily disability.

A blow on the arm with temporary numbness would suggest the idea of paralysis of the limb. This idea, exerting by means of the unconscious motive a strong positive influence, would keep the searchlight of attention fixed upon it; all evidence to the contrary would for a similar reason be excluded from the circle of illumination, so that in a short while the paralysis of the arm became a fixed belief.

Many of the phenomena of daily life depend upon the influence of unconscious motives in directing attention towards certain aspects of self and environment and away from others. Men are notoriously blind to their own faults. They tend to remember their virtues and successes, to forget their failures and peccadilloes. Many acts of forgetting depend upon such a cause.

The man who is being influenced in his actions or his judgment by an unconscious motive is usually ready to defend his position upon logical grounds. His arguments, however plausible, prove on analysis to be fallacious. They are, in fact, further products of the hidden motive, and to distinguish them from processes of true reasoning they are called by psychologists "rationalisations."

#### 4. *Nervous Exhaustion*

Here we are upon uncertain ground. Very little is known of metabolism in the nervous system. The physiologists have shown that the conduction of the nervous impulse can be inhibited by certain poisons—of which alcohol is the classical example—and also by placing the nerve fibre in an atmosphere deprived of oxygen. In clinical practice we are familiar with the effects of acute and chronic intoxication and of anoxæmia upon the higher functions of the nervous system. Under the influence of such causes emotional control is impaired and judgment enfeebled. The individual becomes less capable of purposive thought and action, less able to adapt himself to environmental changes.

The normal capacity for sleep also is disturbed and the resultant insomnia appears to increase the other symptoms.

Excessive or prolonged mental effort appears capable of producing a similar state. If the cause be removed and the individual obtains complete rest, recovery is rapid, but if fatigue has led to insomnia a vicious circle is set up which it is hard to break.

## CLASSIFICATION

1. *The Anxiety Neurosis*

This may be defined as a state of mental distress or anxiety, usually accompanied by physical symptoms, which is out of proportion to the apparent facts of the situation in which it arises. This is the commonest of the neuroses. In the light of the phenomena already described it is easy to see how it may develop from a simple or conditioned emotional reaction. Anxiety gives rise to mental and bodily symptoms. These are misinterpreted by the sufferer, who argues that headaches and lack of concentration denote commencing insanity, palpitation is a sign of heart disease, and dyspepsia of organic disease of the stomach. The original cause of the emotional disturbance being unseen or forgotten, the patient continues to be anxious about his symptoms and so to perpetuate them. These are the neurotics who suffer most from erroneous medical diagnosis. It is true that a confident diagnosis of movable kidney, misplaced uterus, or ileal stasis, with promise of surgical relief, will often relieve symptoms for a time—in so far as any such assurance or procedure will relieve or mitigate anxiety. But, apart from the possible harm done by unnecessary operations, the inevitable recurrence of symptoms undermines the patient's belief in the medical profession. Hence, probably, in some of these cases psychotherapy, when ultimately applied, is more effective in the hands of the quack than the physician.

2. *Hysteria*

Hysteria may be defined as the unconscious assumption of symptoms of illness, either for a particular motive or as the result of suggestion. An example has already been given. Although the relative ætiological importance of unconscious motive and suggestion varies, both are constantly present.

3. *Neurasthenia*

Neurasthenia, which should be clearly distinguished from anxiety neurosis, is a state in which, either from chronic intoxication or prolonged mental strain, the higher functions of the nervous system are impaired, and the individual is therefore rendered incapable of adaptation to the ordinary environmental stress.

Although these three neuroses are clearly distinguishable, they are often combined in the same patient. Thus neurasthenia, weakening the patient's powers of adaptation, renders

him more liable than one in normal health to anxiety neurosis or hysteria.

There is also frequently a hysterical element present in the anxiety neurosis in that the symptoms, which the patient believes are those of organic illness, provide a refuge from some emotional problem.

Further, an anxiety neurosis is likely, if prolonged, to lead to neurasthenia, the mistake commonly made in such a case being to recognise the neurasthenic element but miss the underlying anxiety neurosis.

Of the three neuroses which have been described, the anxiety condition is by far the commonest; hysteria is of less frequent occurrence, and therefore more likely to catch the physician off his guard; neurasthenia, though commonly diagnosed, is, I believe, rare.

Of the first two conditions I have been able to find several examples in the notes of the past few weeks; for the last I have had to search the records of several months before finding a good illustrative case.

#### ILLUSTRATIVE CASES

##### *Anxiety Neurosis*

A married woman aged 26 was brought to see me on account of headaches which she felt convinced were due to a cerebral tumour. The "headaches" were described in terms of discomfort, *i.e.* a feeling as if the head would burst, a feeling of trickling through the head, a rawness on top of the head. With this were associated various fears, and dread of insanity was prominent among them. She had had no previous illness. She had been married two and a half years, but had no children. The illness had commenced three and a half years previously, shortly before her engagement.

The diagnosis of neurasthenia had been made on several occasions, the cause alleged being overwork (before her marriage she worked as a typist). Rest cures had been prescribed without any benefit. The following history was elicited:—

Shortly after she had become secretly engaged, a girl who worked in the same office and had previously had an affair with her fiancé informed her that the man had had venereal disease. The patient was completely ignorant of these matters, but, trusting her lover, told him of this. His denial she at once accepted.

Her informant, however, again came to her with a further and more circumstantial tale, adding that the disease might be conveyed by kissing, and that she (the patient) might have

already acquired it. This story the patient set herself resolutely to disbelieve. But from this time she began to suffer from attacks of panic with physical accompaniments in the shape of headache, dyspepsia and sleeplessness. A month before her marriage she sought medical advice as to the wisdom of embarking upon this venture. She was advised this was no bar and that marriage might do her good. After her wedding she refused to allow the marriage to be consummated on the ground that she was unfit, and that her children might be unfit. Her symptoms at this time became worse. Actually at the end of two and a half years of married life she remained *virgo intacta*.

There was eventually no doubt at all either in my mind or hers that her anxiety was rooted in a dread lest the man she loved had had venereal disease, that she might have acquired it (she had heard of syphilis affecting the brain) and that any child born of the marriage might also be infected. These were solid enough grounds for her state of dread. Later investigation proved that her husband had had gonorrhœa some years before marriage, but had been cured by adequate treatment. Her fears, therefore, were to some extent based upon fact.

Such was the story as it eventually presented itself. When she first came to me, however, although the facts were available from her memory, she was quite unaware that they were in any way related to her symptoms. The dominant note in her mind was loyalty to her husband and implicit belief in his word. Under these circumstances fears rooted in disbelief could not be admitted as such, for they would have spelt disloyalty. The causes of her anxiety stared her in the face yet she could not see them. Her attention was directed elsewhere. She was looking for the opposite of that which was before her eyes. She was, in fact, unconscious of the emotional problems which were the cause of her symptoms. But the symptoms required an explanation. She was told by friends and by medical men that her "nerves" were wrong. She became no better as the result of the treatment prescribed. She began to think that she had some deep-rooted incurable disease. Thus anxiety begot anxiety. The roots of the anxiety being disclosed and removed, her symptoms at once began to clear up.

### *Hysteria*

A single woman aged 44 was admitted to hospital for paraplegia of seven years' duration. The illness was said to have begun with pains in the limbs. She then became unable to walk, and was put to bed. Subsequently she was in several hospitals and underwent a great deal of treatment without benefit. Eventually she was considered incurable.

From the history it appeared that she was one of a large family, moderately well educated, obliged to go out into the world to earn a living at the uncongenial work of nursemaid at the wage of £20 a year. At the age of 37 she became engaged to an officer's batman, a regular soldier, ten years her junior and without any education. That the match was unsuitable was plain to all who knew her, and she was advised by them against it.

The marriage licence having been procured her illness developed. The wedding was then postponed "until she got better." She was in hospital for three months and there received purely physical treatment. She was discharged no better. Similar treatment in other hospitals was equally ineffective. She and her relatives came to the conclusion that she was incurable. Medical advisers hinted darkly at deep-seated mischief in the nervous system.

As the result of psychotherapy this patient was again able to walk. The ultimate prognosis, however, is uncertain since the haven of invalidism lies fatally open to her as a means of escape from life's hardships.

The origin and development of this neurosis may be thus briefly described:—A real physical illness (probably trivial) came at a moment when it provided a convenient excuse for postponing her marriage. This marriage in her heart she knew would be disastrous. Yet it offered the only means of escape from a life of dullness and drudgery. Illness came as a blessing in disguise. It provided her with a reason for postponing the marriage without breaking off the engagement. Her attention turned naturally towards such evidence as supported the diagnosis of organic disease. Medical suggestion played its part. Failure of treatment to effect a cure was accepted by her as proof that the illness would be a long one. As time dragged on the illness itself led to her finding shelter and comfort. The engagement was broken off by the man, but by this time the emotional roots of her trouble were lost to sight. The conviction of incurable disease became as much a reality to her as any physical fact.

#### *Neurasthenia*

A man aged 44 engaged in the dairy business complained that for the past year he had noticed himself increasingly slow and forgetful at his work. For two or three months he had felt muddled, had pains in his heart, discomforts in his throat, a sensation of pressure upon the eyes, a jumping sensation in the left occipital region, and a feeling as if his neck were in a vice. Anything in the nature of an argument upset him so that he could not stand it. Going down steep hills he was afraid of falling.

For some years he had suffered from insomnia. He would be awake for hours thinking about his business.

Investigation of the family history showed that his father had been an unstable person, who deserted his wife and family and had not been heard of since. One brother suffered from epilepsy.

The patient had got on well at school, and had then been employed as a solicitor's clerk, but could not adapt himself to an indoor life, and thus eventually took up dairy work. He had had no physical illness, was a teetotaller, and smoked not more than two ounces of tobacco a week. He had, however, for sixteen years consistently overworked. His day began at 4 a.m. and ended at 9 p.m. with little rest. Saturdays and Sundays were his busiest days, as he had to put his accounts in order during the week-end. During this period of sixteen years he had never had more than a week's holiday.

Treatment of the insomnia, followed by a sea voyage, resulted in improvement, but on returning to work he was again troubled with sleeplessness and many of his old symptoms returned.



# GALL-STONES

## AN ACCOUNT OF THE CASES AT GUY'S HOSPITAL FROM 1895 TO 1915\*

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### *Introduction*

IN a previous communication (*Guy's Hospital Reports*, lxxiv., 1924) it was shown that the late results of cholecystectomy were better than those of cholecystostomy. It is now proposed to give an account of all cases of gall-stones admitted to Guy's Hospital during the years 1895 to 1915. The year 1895 marks the beginning of gall-stone surgery at Guy's Hospital, and in the following twenty years there was the transition from antiseptic to aseptic surgery. It may be objected that the material is old and that now the results are much better. An investigation of the cases from 1916 onwards will prove or disprove that. There is much to be learnt from the practice of the pioneers of any branch of surgery. In the light of experience with reflection and meditation what may appear to the pioneers themselves or to their critics to have been surgical crimes were perhaps committed. Practical experience of the difficulties which may be met with in the surgery of the biliary apparatus will convince the surgeon of to-day that even in the heat of a struggle the pioneers showed a great resource, combined with judgment, discretion and prudence.

### *The Material*

The four hundred and twenty-five cases (318 women and 107 men) may be grouped as in Table I.

### *The Yearly Admissions*

These are shown in Table II (1, 2, 5 and 6, Table I).

\* Part of the expense of this research was defrayed by a grant from the Clinical Research Committee, Guy's Hospital.

TABLE I.

	Total.	Men.	Women.
1. Gall-stones, operation, recovery . . . .	278	61	217
2. Gall-stones, operation, death . . . .	35	11	24
3. Operation, no gall-stones found, recovery . .	16	5	11
4. Operation, no gall-stones found, died . . .	2	0	2
5. No operation but diagnosis confirmed by passage of stones . . . . .	18	5	13
6. Gall-stones, no operation, death . . . .	12	3	9
7. Gall-stones found post-mortem in patients dying from other disease . . . . .	30	15	15
8. Carcinoma of the gall bladder or ducts with gall-stones found at operation . . . . .	9	1	8
9. Carcinoma of the gall bladder or ducts with gall-stones found post-mortem . . . . .	13	5	8
10. Carcinoma of pancreas with gall-stones found at operation . . . . .	7	0	7
11. Carcinoma of pancreas with gall-stones found post-mortem . . . . .	2	1	1
12. Gall-stones impacted in the ileum, operation, death . . . . .	3	0	3
	425	107	318

TABLE II.

Year.	Men.	Women.	Total.	Year.	Men.	Women.	Total.
1895	1	1	2	1906	4	8	12
1896	0	5	5	1907	3	17	20
1897	2	11	13	1908	1	17	18
1898	0	8	8	1909	4	16	20
1899	4	3	7	1910	2	18	20
1900	3	5	8	1911	6	34	40
1901	3	9	12	1912	15	23	38
1902	2	10	12	1913	6	19	25
1903	1	6	7	1914	10	17	27
1904	5	13	18	1915	8	15	23
1905	0	8	8				
	21	79	100		59	184	243

It will be seen that in the second decade the number of men admitted was nearly trebled and the number of women increased nearly two and a half times. The factors leading to this appear to have been: a growing confidence in operations on the abdomen on the part of patients and their medical advisers; the recognition of the value of surgery in the treatment of gall-stones. It must, however, be admitted that surgical treatment was usually undertaken for the complications. Operation for the early symptoms or as a result of the investigation of a case of "dyspepsia" was rare even in the later years.

*The Age Incidence*

This is shown in Table III (1, 2, 5 and 6, Table I).

TABLE III.

Age.	Men.	Women.	Total.
21-30	4	22	26
31-40	6	61	67
41-50	36	73	109
51-60	21	66	87
61-70	12	34	46
71-80	1	7	8
	80 (23%)	263 (77%)	343

The figures support the conclusions in the previous communication (*loc. cit.*).

*Age Incidence and Operative Mortality*

This is shown in Table IV (1 and 2, Table I).

TABLE IV.

Age.	Men.			Women.			Total mortality.
	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.	
21-30	4	0	0	19	0	0	0%
31-40	5	1	20	58	3	5	6%
41-50	33	3	9	68	4	5.8	6.9%
51-60	19	2	10.5	59	5	8.5	9%
61-70	10	4	40	31	11	35.5	36.6%
71-80	1	1	100	6	1	16.72	28.5%
	72	11	15	241	24	9.9	11.2%

The chief points of interest are the greater mortality in men and the great increase of post-operative deaths after the age of sixty.

*The Operation Performed*

This is shown in Table V.

It will be seen that the mortality of cholecystostomy and cholecystectomy were about equal. The mortality of cholecystectomy would have been much higher than cholecystostomy but for a wise and judicious selection of the cases submitted to cholecystectomy. On the other hand, cholecystostomy is the minimal surgical procedure that can be adopted and therefore includes the more serious operative risks.

It will be seen later that the high operative mortality of cholecystostomy with choledochotomy and choledochotomy is

TABLE V.

Operation performed.	No. of cases.	No. of deaths.	Percentage mortality.
Cholecystendysis . . . . .	10	0	0
Cholecystostomy . . . . .	176	16	9.5
Cholecystectomy . . . . .	77	7	9
Cholecystostomy and Choledochotomy . . . . .	28	9	32
Cholecystectomy and Choledochotomy . . . . .	10	0	0
Choledochotomy . . . . .	10	3	30
	311	35	11.2

*Note.*—In two cases the biliary apparatus was not opened.

due to failure to relieve the complications of gall-stones present before operation rather than to the operation itself.

The same judicious selection of cases is apparent in the ten operations of cholecystectomy with choledochotomy without a death. Judging from the immediate and late results, this is the ideal operation for gall-stones when the disease involves the common duct in addition to the gall bladder. At the same time the late results of cholecystostomy with choledochotomy are sufficiently good to justify its performance in preference to taking the greater risk of removing the gall bladder as a routine. In these cases the gall bladder does not resemble an acutely inflamed or gangrenous appendix lying free in the peritoneal cavity. The gall bladder is chronically inflamed, contracted, lying in a mass of adhesions, possibly surrounded by an abscess, and bleeding is increased by jaundice. The comparison must therefore be made with an appendix after the fourth day of an attack of inflammation, firmly fixed in adhesions at the back of the abdominal cavity. A routine removal of either organ carries a risk of death from peritonitis. Further, the elderly patient with gall-stones may die from a very low form of peritonitis due to a lack of peritoneal resistance.

#### *The Operative Mortality in Four Periods*

This is shown in Table VI.

TABLE VI.

Period: Years.	Chole- cystendysis.			Chole- cystostomy.			Chole- cystectomy.			Chole- cystostomy and Chole- dochotomy.			Chole- cystectomy and Chole- dochotomy.			Chole- dochotomy.		
	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.	No. of cases.	No. of deaths.	Percentage mortality.
1895-1900	3	0	0	23	6	26	1	1	100	3	1	33	0	0	0	3	1	33
1901-1905	4	0	0	8	1	12.5	12	2	16.7	3	0	0	2	0	0	3	2	66
1906-1910	2	0	0	45	3	6.7	12	0	0	12	2	16.7	4	0	0	0	0	0
1911-1915	1	0	0	100	6	6	12	2	16.7	15	6	40	4	0	0	12	0	0

This is interesting because it shows that the mortality of cholecystostomy was reduced to six per cent. in the last ten years, whereas the mortality of cholecystectomy remained the same for fifteen years.

It is often alleged in literature that British surgeons have neglected to remove the gall bladder in suitable cases from conservative motives. This allegation is untrue, for from 1901 to 1905 there were three times as many cholecystectomies as cholecystostomies. During that period the selective mortality of cholecystectomy was minimum, but the general mortality of cholecystostomy was twice that of the subsequent ten years.

It will be seen also that the number of cholecystectomies in each period of five years has not materially increased, whereas there was an enormous increase of cholecystostomies. It must be concluded that even in the last five years (1911-1915) the cases were not seen early enough to justify cholecystectomy.

It is a noteworthy and important fact that those surgeons who made a thorough and routine examination of the right and left hepatic ducts and the common bile duct with the scoop were prone to do cholecystostomy rather than cholecystectomy. From the point of view of the immediate prognosis this was justified, for it was the condition of the common bile duct which dominated the issue, and quite frequently stones were found which otherwise would have been missed. On the other hand, where the surgeon was satisfied from external palpation that the common duct did not contain stones there was a tendency to do cholecystectomy. The risks of removing contracted adherent gall bladders are very great. A contracted and adherent gall bladder is presumptive evidence of disease in the common duct. Removal of such a gall bladder should only be done at the end of the operation after the ducts have been thoroughly explored and a tube has been sewn into the common duct. Frequently it will be found that the division of adhesions to expose and thoroughly explore the common duct has been so extensive that removal of the gall bladder will add little to the immediate risk. If removal of the gall bladder requires still further division of adhesions and the patient is jaundiced, there must be reflection and consideration of the risks.

#### *The Operative Mortality in Age Periods*

This is shown in Table VII, A, B and C.

Table VII shows the influence of age and sex on the operative mortality. It is interesting to note the low mortality of cholecystostomy up to the age of fifty, figures which compare favourably with statistics from any part of the world, and also

TABLE VII.

**A. MEN.**

Age.	Cholecystostomy.			Cholecystectomy.			Cholecystectomy and Cholecholethotomy.			Cholecholethotomy.			Nil Done.
	No. of cases.	No. of deaths.	Per-centage mor-tality.	No. of cases.	No. of deaths.	Per-centage mor-tality.	No. of cases.	No. of deaths.	Per-centage mor-tality.	No. of cases.	No. of deaths.	Per-centage mor-tality.	
21-30	0	0	0	3	0	0	0	0	0	0	0	0	0
31-40	0	0	0	4	0	0	0	1	100	0	0	0	0
41-50	1	0	0	19	1	5	4	1	25	0	3	33	0
51-60	0	0	0	13	1	7·6	2	0	0	3	1	33	0
61-70	1	0	0	7	4	55	0	0	0	0	0	0	0
71-80	0	0	0	1	1	100	0	0	0	0	0	0	0

## B. Women.

[illegible]

### C (A + B). MEN AND WOMEN.

[illegible]

the low mortality of cholecystectomy between fifty and sixty by careful selection of suitable cases.

The influence of age on the mortality of cholecystostomy with choledochotomy is also shown.

*The Average Time in Hospital*

This is shown in Table VIII, A and B.

TABLE VIII.

A.

Operation.	No. of cases.	Average time in hospital.
Cholecystendysis . . . . .	10	23.5 days
Cholecystostomy . . . . .	160	38 "
Cholecystectomy . . . . .	70	22.3 "
Cholecystostomy with Choledochotomy . . . . .	19	32.8 "
Cholecystectomy with Choledochotomy . . . . .	10	33.3 "
Choledochotomy . . . . .	7	46.8 "

B. In four periods.

Period.	Cholecystendysis.		Cholecystostomy.		Cholecystectomy.		Cholecystostomy and Choledochotomy.		Cholecystectomy and Choledochotomy.		Choledochotomy.	
	No. of cases.	Average time in days.	No. of cases.	Average time in days.	No. of cases.	Average time in days.	No. of cases.	Average time in days.	No. of cases.	Average time in days.	No. of cases.	Average time in days.
1895-1900	3	42.7	17	53	0	0	2	33	0	0	2	66
1901-1905	4	17	7	48	24	33	3	43	2	33.5	1	42
1906-1910	2	18	42	39	20	24.5	5	40	4	33	1	?
1911-1915	1	20	94	33	26	23	9	25	4	27	3	30

The most significant feature in Table VIII is the more rapid closing of the cholecystostomy fistula in the later years because the rubber tube was invaginated into the gall bladder and because the gall bladder was not sutured to the parietal peritoneum or abdominal wall.

In the earlier years the protective powers of the peritoneum were unknown. The peritoneum was regarded more as an enemy than as a friend and the gall bladder was sutured to the abdominal wall so as to be extraperitoneal before it was opened. The operation of cholecystendysis was also done from fear of infecting the peritoneum.

*The Incision Used*

This is shown in Table IX.

TABLE IX.

No. of cases.	Incision.		
	Vertical.	Oblique.	Vertical and Transverse.
278 (operation, recovery)	235	37	6
35 (operation, death)	29	3 (one because vertical did not give adequate exposure)	3 (in two of these vertical did not give adequate exposure)

A consideration of the incision used shows that the transverse or oblique incision gives the best exposure of the biliary apparatus.

*The Difficulties found at Operation*

These are summarised in Table X.

TABLE X.

No. of cases.	Difficulties.							
	Fistula of common duct.	Fistula of gall bladder.	Hæmorrhage.	Abscess round gall bladder.	Adhesions.	Enlarged gall bladder.	Small gall bladder.	Gall bladder not examined.
278 (operation, recovery)	1	2	5	5	54	61	18	0
35 (operation, death)		2*	6	9	21	10	17	1

Table X shows the greater proportion of difficulties in the fatal cases. The frequency of hæmorrhage, abscess round the gall bladder, adhesions and contracted gall bladder shows the limitations of cholecystectomy in advanced disease of the biliary apparatus.

Some of the difficulties met with in patients who recovered are worthy of mention :—

(a) Common duct adherent to the stomach, hole in the stomach closed, one inch of the common duct resected and the ends sutured—uncomplicated recovery.

(b) Transverse colon opened and sutured.

\* 1 Duodenum, 1 hepatic flexure of the colon.



- (c) Cholecysto-gastric fistula.
- (d) Forceps left on cystic artery for forty-eight hours.
- (e) Stone in common bile duct pushed into duodenum.
- (f) Retro-duodenal choledochotomy.
- (g) Abscess round gall bladder first drained and gall bladder explored later.
- (h) Difficulty in removing stones from the cystic duct—in one case the stone was left in the duct and removed following repeated injections of olive oil into the gall bladder through the drainage tube.
- (i) Gall bladder not touched and choledochotomy.
- (j) Multiple small abscesses in the liver.



FIG. A.

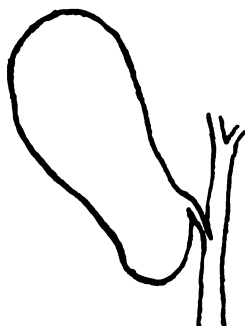


FIG. B.

(k) Stones not found, gall bladder drained and stones removed later.

(l) Stone in diverticulum of gall bladder (Fig. A). This must be distinguished from the boss-like projection which forms near the neck of a distended gall bladder and obscures the cystic duct and common bile duct (Fig. B).

(m) Obstruction of common bile duct by blood clot; operation, clot turned out, choledochotomy—recovery.

### *The Signs present at Operation*

Certain of these are summarised in Table XI.

TABLE XI.

No. of cases in which these signs were noted as being present.	Signs.			
	Jaundice.	Clay stools.	Bile in urine.	Repeated rigors.
178	63	14	37	4
(operation, recovery)				
35	26	9	12	7
(operation, death)				

Table XI shows the effect of biliary obstruction and infection on the prognosis. A case of gall-stones is not such an urgent

surgical emergency as the early stages of an attack of acute appendicitis. The advantage of delaying operation until jaundice has diminished or disappeared is shown in the cases under review. There are cases of cholangitis which recovered without early operation and others where a timely operation because of repeated rigors and deepening jaundice prevented the onset of cholæmia. In the eight fatal cases (Table XV) an expectant attitude was adopted at first. Excluding three cases where stones were left in the common duct a constant feature is delay in drainage of bile through the cholecystostomy tube, so that it is evident that the common bile duct must be drained in these cases. Further, there is evidence that bile does not drain freely into the duodenum. This may be met by putting a small rubber tube into the common bile duct and pushing the lower end through the ampulla into the duodenum—a method I have employed successfully on two occasions.

*The Distribution of the Gall-Stones*

This is shown in Table XII.

TABLE XII.

No. of cases.	Distribution of the Stones.				
	Gall bladder only.	Gall bladder and cystic duct only.	Cystic duct only.	Gall bladder, cystic duct and common bile duct.	Common bile duct only.
278 (operation, recovery)	183	23	23	36	13
35 (operation, death)	17	3	1	11	3
12 (no operation, death)	7	0	0	3	2
Stones found post-mortem in five cases in which death followed operation.	1	0	0	0	4
325	208	26	24	50	22

Table XII shows two factors which increase the mortality of operations for gall-stones: spread of infection through the wall of the gall bladder and spread of infection and stones in the common bile and hepatic ducts. With earlier diagnosis and operation this would be rare instead of so common.

*The Complications in the Cases which Recovered*

These are summarised in Table XIII.

TABLE XIII.

Fistula . . . . .	26 cases
Sepsis, drainage . . . . .	5 „
Femoral thrombosis . . . . .	2 „
Empyæma and subdiaphragmatic abscess, drained . . . . .	1 case
Second operation . . . . .	14 cases
(Stones found in gall bladder in eight and in common bile duct in six.)	
Third operation . . . . .	2 cases
Seven operations . . . . .	1 case

**Fistula.**—Suture of the gall bladder to the abdominal wall as a cause of fistula has already been considered. The other causes were obliteration of the cystic duct and unrelieved obstruction in the common duct.

**Femoral Thrombosis.**—This is an interesting post-operative complication because the thrombosis of the left femoral vein was present in patients who were jaundiced.

The question of secondary operations was dealt with in *Guy's Hospital Reports*, Vol. lxxiv. No. 3, Series 4.

### *The Cause of Death*

This is shown in Table XIV.

TABLE XIV.

In thirty-five cases in which death followed operation.	In twelve cases in which no operation was performed.
Cholangitis . . . . . 8	Cholangitis . . . . . 6
Pneumonia . . . . . 6	Abscess of Lung . . . . . 1
Diffuse Peritonitis . . . . . 4	Subphrenic Abscess . . . . . 3
Hæmorrhage . . . . . 4	Suppurative Pericarditis . . . . . 1
Subphrenic Abscess . . . . . 3	Empyæma of Gall Bladder . . . . . 1
Post-operative Shock . . . . . 2	
Cardiac Failure . . . . . 2	
Acute Hæmorrhagic Pancreatitis . . . . . 2	
Hæmatemesis . . . . . 2	
Pyloric Stenosis . . . . . 1	
Perforated Duodenal Ulcer . . . . . 1	
35	12

In Table XIV is shown the cause of death with and without operation.

The deaths from cholangitis after operation were due to failure to relieve the condition and were not due to the operation. In particular must be mentioned delay in drainage of bile through a cholecystostomy fistula and into the duodenum. In three cases stones were left in the hepatic and common bile ducts. In the other cases the operation as a factor in causing death needs further consideration.

### *The Deaths from Cholangitis following Operation*

These cases are summarised in Table XV.

TABLE XV.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rigors.					
1 1896	32	—	Yes.	One day after admission.	Yes.	No.	No.	No.	Cholecystostomy; no drainage of bile after operation.	In common duct only.	Contracted gall bladder.	Cholangitis and pyelephlebitis. Three days after operation.	Nil.
2 1901	53	Yes.	—	Seven days after admission.	Yes.	Yes.	Yes.	Yes.	Cholechoctomy.	In common duct only. Free drainage of bile at once.	Gall bladder was not examined because it was contracted and buried in adhesions.	Cholemia. Eight days after operation.	Nil.
3 1902	60	—	Yes.	Three days after admission.	Yes.	—	Yes.	—	Cholechoctomy.	In gall bladder, cystic duct and common duct.	Contracted gall bladder.	Suppurative cholangitis. Two days after operation.	Nil.
4 1904	56	—	Yes.	Seven days after admission.	—	—	—	—	Cholecystostomy; no drainage of bile for two days.	In gall bladder.	Contracted gall bladder and adhesions.	Cholangitis. Three days after operation.	Four stones in common bile duct.
5 1908	62	—	Yes.	Two days after admission.	Yes.	Yes.	Yes.	Yes.	Cholecystostomy.	In gall bladder.	Contracted gall bladder and adhesions. Vertical incision followed by transverse.	Suppurative cholangitis. Thirty-six hours after operation.	Many stones in common bile duct and right and left hepatic ducts.
6 1908	65	—	Yes.	One day after admission.	Yes.	Yes.	Yes.	Yes.	Cholecystostomy and cholechoctomy.	In common bile duct only.	Hæmorrhage. Adhesions. Contracted gall bladder.	Cholemia. hours after operation. A few hours after operation.	Twelve stones in common bile duct and hepatic ducts.
7 1914	36	Yes.	—	One day after admission.	Yes.	—	—	Yes.	Cholecystostomy and cholechoctomy.	In gall bladder, cystic duct and common bile duct.	Abscess round contracted gall bladder. Adhesions.	Suppurative cholangitis. Eleven days after operation. The wound sloughed and broke down and was explored again on day before death.	Nil.
8 1914	71	—	Yes.	Three days after admission.	Yes.	Yes.	Yes.	—	Cholecystostomy.	In gall bladder and cystic duct (115).	Contracted gall bladder.	Cholemia. Nine days after operation.	Nil.

The points to note are that two patients were under forty, the distribution of the stones, expectant treatment, contracted gall bladders, adhesions, jaundice, hæmorrhage, abscess round the gall bladder and stones left in common bile and hepatic ducts.

*The Deaths from Pneumonia following Operation*

These are summarised in Table XVI.

There is no reason to suppose that the anæsthetic was the sole cause of the pneumonia. Post-anæsthetic pneumonia was surprisingly rare in this series. The patients on whom cholecystectomy was done were younger than the others. It is reasonable to suppose that cholecystostomy would have been a safer operation in Case 2.

*The Deaths from Diffuse Peritonitis following Operation*

These are summarised in Table XVII.

There was jaundice and an abscess round the gall bladder in the fatal case of cholecystectomy. In the fatal case of cholecystectomy, the peritonitis arose after operation from division of adhesions.

*The Deaths from Hæmorrhage following Operation*

These are summarised in Table XVIII.

Note that there were no deaths from hæmorrhage following cholecystostomy.

The death following cholecystectomy was due to hæmorrhage from the cystic artery.

All the patients were jaundiced.

*The Deaths from Subphrenic Abscess following Operation*

These are summarised in Table XIX.

There was no death from subphrenic abscess after 1900 because of the adoption of the Fowler position.

There was an abscess round the gall bladder and death was due to spread of infection.

*The Deaths from Post-operative Shock and Heart Failure following Operation*

These are summarised in Table XX.

The points to notice are the ages of the patients; the presence of adhesions round the gall bladder in Case 1 and of an abscess round the gall bladder in Cases 2, 3 and 4; the higher mortality of cholecystectomy due directly to the operation.

TABLE XVI.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Hicors.					
1 1895	68	Yes.	—	Two days after ad-mission.	—	—	—	—	Cholecystostomy.	In gall bladder only.	Contracted gall bladder.	Pneumonia. Two days after operation.	Nil.
2 1902	60	—	Yes.	Eight days after ad-mission.	Yes.	Yes.	Yes.	—	Cholecystectomy.	In gall bladder, cystic duct and common bile duct.	Contracted gall bladder.	Pneumonia. Eleven days after operation.	Nil.
3 1909	74	Yes.	—	Two days after ad-mission.	Yes.	Yes.	—	—	Cholecystostomy.	In gall bladder and cystic duct.	Contracted gall bladder.	Pneumonia. Two days after operation.	Nil.
4 1910	50	—	Yes.	One day after ad-mission.	Yes.	—	—	Yes.	Cholecystectomy.	In gall bladder only.	Enlarged gall bladder.	Pneumonia. Operation. Gangrene of lung. Nine days after operation.	Nil.
5 1912	62	—	Yes.	Four days after ad-mission.	Yes.	—	—	Yes.	Cholecystostomy with choledochotomy.	In gall bladder, cystic duct and common bile duct.	Enlarged gall bladder. Adhesions.	Pneumonia. Eighteen days after operation.	Nil.
1914	66	—	Yes.	One day after ad-mission.	Yes.	—	—	—	Cholecystostomy.	In gall bladder.	Enlarged gall bladder. Adhesions.	Pneumonia. Five days after operation.	Nil.

TABLE XVII.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rigors.					
1	1896	—	Yes.	One day after admission.	Yes.	—	—	—	Cholecystostomy.	In gall bladder.	Abscess round an enlarged gall bladder.	Diffuse peritonitis. Three days after operation.	Nil.
2	1908	—	Yes.	Twenty-seven days after admission.	—	—	—	—	Cholecystectomy.	In gall bladder.	Adhesions. Enlarged gall bladder.	Diffuse peritonitis. Two days after operation.	Nil.
3	1908	—	Yes.	Five days after admission.	Yes.	Yes.	—	—	Cholecystostomy and choledochotomy.	In gall bladder, cystic duct and common bile duct.	Adhesions. Fistula between gall bladder and duodenum.	Diffuse peritonitis. Five days after operation.	Nil.
4	1913	Yes.	—	Thirteen days after admission.	Yes.	Yes.	Yes.	—	Cholecystostomy and choledochotomy.	In gall bladder, cystic duct and common bile duct.	Hæmorrhage. Adhesions. Contracted gall bladder.	Diffuse peritonitis. Twelve days after operation.	Nil.

TABLE XVIII.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rigors.					
1	1897	—	Yes.	?	Yes.	—	—	—	Cholecystectomy.	Gall bladder only.	Hæmorrhage. Adhesions.	Hæmorrhage. Five days after operation.	Nil.
2	1898	—	Yes.	Five days after admission.	Yes.	Yes.	Yes.	—	Cholecystostomy and Choledochotomy.	Gall bladder, cystic duct and common bile duct.	Hæmorrhage.	Hæmorrhage. Twelve hours after operation.	In common duct
3	1913	—	Yes.	One day after admission.	Yes.	Yes.	Yes.	—	Cholecystostomy and Choledochotomy.	Gall bladder; cystic duct and common bile duct.	Abscess, round contracted gall bladder. Adhesions.	Rupture of common bile duct into lesser sac. Secondary hæmorrhage. Six days after operation.	Nil.
4	1913	—	Yes.	Four days after admission.	Yes.	—	—	—	Cholecystostomy and Choledochotomy.	Gall bladder, cystic duct and common bile duct.	Hæmorrhage. Adhesions. Contracted gall bladder.	Hæmorrhage. Five hours after operation.	Nil.



TABLE XIX.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rhizors.					
1	1896	—	Yes.	One day after admission.	Yes.	—	—	—	Cholecystostomy.	In gall bladder.	Abscess round gall bladder. Adhesions.	Subphrenic abscess. Two days after operation.	Nil.
2	1896	—	Yes.	One day after admission.	Yes.	—	—	—	Cholecystostomy.	In gall bladder.	Abscess round contracted gall bladder. Adhesions.	Subphrenic abscess. Sixteen days after operation.	Nil.
3	1900	Yes.	—	On day of admission.	—	—	—	—	Cholecystostomy.	In gall bladder.	Gangrenous gall bladder. Abscess round gall bladder.	Subphrenic abscess. Three days after operation.	Nil.

TABLE XX.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rigors.					
1 1902	67	—	Yes.	Four days after admission.	—	—	—	—	Cholecystectomy.	In gall bladder.	Adhesions. Contracted gall bladder.	Cardiac failure. Seven days after operation.	Nil.
2 1911	62	—	Yes.	Three days after admission.	Yes.	—	—	—	Cholecystectomy.	In gall bladder.	Abscess an enlarged gall bladder.	Cardiac failure. Four days after operation.	Nil.
3 1912	56	Yes.	—	On day of admission.	—	—	—	—	Cholecystostomy.	In gall bladder.	Adhesions. Abscess round gall bladder.	Post-operative shock on day following operation.	Nil.
4 1914	67	Yes.	—	On day of admission.	—	—	—	—	Cholecystostomy.	In gall bladder.	Abscess round gall bladder. Adhesions. Ulceration of gall bladder into hepatic flexure of colon. Peritonitis.	Post-operative shock one hour after operation.	In gall bladder.

## GALL-STONES

TABLE XXI.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
					Faun- dice.	Bile in urine.	Clay stools.	Iligors.					
1	1899	46	Men.	—	Yes.	Yes.	—	Yes.	Cholecystostomy. (Cholecystostomy one year before without relief from symptoms.)	In common bile duct.	Adhesions. Contracted gall bladder.	Acute pancreatitis. Fat necrosis. Ten days after operation.	Nil.
2	1912	65	Yes.	—	Yes.	—	—	—	Cholecystostomy.	In gall bladder, cystic duct and common bile duct.	Ulceration of stone from common bile duct into pancreas. Acute haemorrhagic pancreatitis.	Acute haemorrhagic pancreatitis. Fat necrosis. One day after operation.	Nil.

TABLE XXII.

Year.	Age.	Sex.		When operation was performed.	Signs.				Nature of operation performed.	Distribution of gall-stones.	Difficulties at operation.	Cause of death.	Stones found post-mortem.
		Men.	Women.		Jaundice.	Bile in urine.	Clay stools.	Rigors.					
1 1909	70	Yes.	—	On day of admission.	—	—	—	—	Cholecystostomy.	In gall bladder.	Hæmorrhage. Adhesions. Enlarged gall bladder. Adhesions.	Pyloric stenosis. Six days after operation. Perforated acute ulcer of the duodenum. Perforated on day of death, six days after first operation. Died thirty-two days after operation. Suppuration in wound. Hæmatemesis from multiple acute ulcers in the stomach.	Nil.
2 1911	60	—	Yes.	One day after admission.	Yes.	Yes.	—	Yes.	Cholecystostomy.	In gall bladder.			Nil.
3 1911	45	—	Yes.	Eight days after admission.	—	—	—	—	Cholecystectomy.	In gall bladder and cystic duct.	Enlarged gall bladder.		Nil.
4 1912	61	—	Yes.	One day after admission.	Yes.	—	—	—	Cholecystostomy and Choledochotomy.	In gall bladder, cystic duct and common bile duct.	Enlarged gall bladder.	Died nine days after operation. Chronic gastric ulcer. Hæmatemesis. Intestines full of blood.	Nil.

*The Deaths from Acute Pancreatitis following Operation*

These are summarised in Table XXI.

These deaths, both in men, were due directly to the failure of operation to relieve a complication of gall-stones. It should be noted that a cholecystostomy was performed on Case 1 a year before the second operation.

*The Deaths from Gastric Ulcer, Duodenal Ulcer and Hæmatemesis*

These are summarised in Table XXII.

In Cases 1 and 4 death was due to a coexisting disease in the stomach. The manipulations at operation may have activated the ulcer and caused hæmorrhage in Case 4.

Case 2 died from perforation of an acute duodenal ulcer which may have been due to pressure of a drainage tube, six days after operation.

The hæmatemesis in Case 3 was due to acute ulceration of the stomach resulting from the suppuration in the wound. A similar complication may occur after suture of a perforated gastric or duodenal ulcer.

*Fatal Cases*

1. Those in which the gross disease was confined to the gall bladder and cystic duct and without jaundice.

1. 1895. Male, 68. *Cholecystostomy*.—Oblique incision made because vertical did not give good access to the biliary apparatus. Contracted gall bladder. Stones in gall bladder. Death two days after operation from pneumonia.
2. 1908. Woman, 64. *Cholecystectomy*.—Adhesions; enlarged gall bladder. Stones in gall bladder. Death two days after operation from peritonitis.
3. 1902. Woman, 67. *Cholecystectomy*.—Adhesions; small gall bladder. Stones in gall bladder. Death from heart failure seven days after operation.
4. 1911. Woman, 45. *Cholecystectomy*. Enlarged gall bladder. Suppuration in the wound. Stones in gall bladder, cystic duct. Death from hæmatemesis thirty-two days after operation.
5. 1909. Woman, 70. *Cholecystostomy*.—Adhesions; enlarged gall bladder. Stones in gall bladder. Death from pyloric stenosis six days after operation.

This shows the excellent results of cholecystostomy when the disease is confined to the gall bladder. There were only two

deaths, and these in patients sixty-eight and seventy years old. In a smaller series of cholecystectomies there were three deaths. The mortality of cholecystectomy is much higher than cholecystostomy. Two of the deaths from cholecystectomy were due to local causes resulting from operation, whereas the deaths following cholecystostomy were due to pneumonia and pyloric stenosis in patients sixty-eight and seventy.

2. Cases in which the gall-stones were confined to the gall bladder and cystic duct, but signs of infection and obstruction of the common duct (jaundice, bile in urine, clay stools and rigors) were present.

1. 1910. Woman, 50. *Cholecystectomy*.—Enlarged gall bladder; jaundice; rigors. Death nine days after operation from pneumonia and gangrene of the lung.
2. 1914. Woman, 66. *Cholecystostomy*.—Enlarged gall bladder; adhesions; jaundice. Death five days after operation from pneumonia.
3. 1909. Man, 74. *Cholecystostomy*.—Small gall bladder; jaundice. Death two days after operation from pneumonia.
4. 1897. Woman, 38. *Cholecystectomy*.—Jaundice; adhesions; hæmorrhage, small gall bladder. Death five days after operation from hæmorrhage.

The chief points to notice are the ages of the fatal cases of cholecystectomy. The death-rate of the selective operation of cholecystectomy was much higher than the general operation of cholecystostomy.

3. Cases in which the stones were confined to the gall bladder and cystic duct with abscess round the gall bladder.

1. 1896. Woman, 63. *Cholecystostomy*.—Jaundice, abscess round an enlarged gall bladder. Death three days after operation from diffuse peritonitis.
2. 1896. Woman, 60. *Cholecystostomy*.—Jaundice; abscess round gall bladder; adhesions. Subphrenic abscess drained. Death two days after operation.
3. 1896. Woman, 45. *Cholecystostomy*.—Jaundice; abscess round contracted gall bladder; adhesions. Death from subphrenic abscess fifteen days after operation.
4. 1900. Man, 50. *Cholecystostomy*.—Abscess round gangrenous gall bladder. Death from subphrenic abscess three days after operation.
5. 1912. Woman, 56. *Cholecystostomy*.—Abscess round gall bladder. Death next day from post-operative shock.

6. 1914. Woman, 67. *Cholecystostomy*.—Abscess and adhesions round gall bladder; ulceration of gall bladder into hepatic flexure of colon; peritonitis. Death from post-operative shock one hour after operation. Stones found in gall bladder post-mortem.
7. 1911. Woman, 62. *Cholecystectomy*.—Jaundice; abscess and adhesions round an enlarged gall bladder. Death from heart failure four days after operation.

Next to complications within the common bile and hepatic ducts, an abscess round the gall bladder is the most common cause of death. Cholecystectomy was out of the question in these cases. The breaking down of adhesions opens up lymphatic spaces and leads to spread of the infection. Where advisable an acutely inflamed or gangrenous gall bladder should be removed, but there are instances in the series under review where gangrenous gall bladders were drained with recovery. Further, the practice of first draining the abscess round the gall bladder and exploring or removing the gall bladder when the acute infection has subsided has much to commend it, because it has saved life. This is a true parallel to the late appendix with adhesions. Cholecystectomy will not save life, and a frantic effort to remove the gall bladder because of the better late results is not justified. The mortality of two or more operations on the biliary apparatus is not so great as to allow the taking of risks at the first operation.

4. Stones present in the common bile duct with signs of obstruction.

1. 1908. Woman, 65. *Cholecystostomy and Choledochotomy*.—Stones in common bile duct; small gall bladder surrounded by adhesions; jaundice; bile in urine; clay stools; rigors. Hæmorrhage at operation. Death same day from cholæmia. Twelve stones found post-mortem in hepatic and common bile ducts.
2. 1914. Man, 36. *Cholecystostomy and Choledochotomy*.—Abscess and adhesions round small gall bladder; jaundice; rigors. Death from suppurative cholangitis eleven days after operation.
3. 1914. Woman, 71. *Cholecystostomy*.—Contracted gall bladder; jaundice; bile in urine; clay stools. Death from cholæmia nine days after operation.
4. 1896. Woman, 32. *Cholecystostomy*.—Jaundice; contracted gall bladder. Death three days after operation from cholangitis and pylephlebitis.

5. 1901. Man, 53. *Choledochotomy*.—Abscess round a small gall bladder which was not examined because it was buried in adhesions. Jaundice; bile in urine; clay stools; rigors. Death eight days after operation from cholæmia.
6. 1902. Woman, 60. *Choledochotomy*.—Adhesions round a small gall bladder; jaundice; clay stools. Death from suppurative cholangitis two days after operation.
7. 1908. Woman, 62. *Cholecystostomy*.—Adhesions; small gall bladder; jaundice; bile in urine; clay stools; rigors. Death thirty-six hours later from suppurative cholangitis. Many stones found in hepatic and common bile ducts post-mortem.
8. 1904. Woman, 56. *Cholecystostomy*.—Adhesions round a small gall bladder. Death from cholangitis three days after operation. Four stones found in common bile duct post-mortem.
9. 1902. Woman, 60. *Cholecystectomy*.—Small gall bladder; jaundice; bile in urine; clay stools. Death eleven days after operation from pneumonia.
10. 1912. Woman, 62. *Cholecystostomy and Choledochotomy*.—Adhesions; enlarged gall bladder; jaundice and rigors. Death from pneumonia eighteen days after operation.
11. 1913. Man, 44. *Cholecystostomy and Choledochotomy*.—Jaundice; bile in urine; clay stools; adhesions; small gall bladder. Hæmorrhage during the operation. Death twelve days later from diffuse peritonitis.
12. 1908. Woman, 63. *Cholecystostomy and Choledochotomy*.—Jaundice; bile in urine; fistula of gall bladder into duodenum. Death five days after operation from diffuse peritonitis.
13. 1913. Woman, 49. *Cholecystostomy and Choledochotomy*.—Jaundice; adhesions; small gall bladder. Hæmorrhage during the operation. Stone left in common bile duct at previous operation—cholecystostomy. Death from hæmorrhage five hours after operation.
14. 1898. Woman, 40. *Cholecystostomy and Choledochotomy*.—Jaundice; bile in urine; clay stools. Hæmorrhage during operation. Death from hæmorrhage twelve hours after operation. Stones found in common duct post-mortem.
15. 1913. Woman, 62. *Cholecystostomy and Choledochotomy*.—Jaundice; bile in urine; clay stools; abscess, and adhesions round a contracted gall bladder.



Death from secondary hæmorrhage six days after operation. Rupture of common bile duct into lesser sac of peritoneum.

16. 1897. Woman, 38. *Cholecystectomy*.—Jaundice; adhesions; enlarged gall bladder. Hæmorrhage during operation. Death from hæmorrhage five days after operation.
17. 1899. Man, 46. *Choledochotomy*.—Cholecystostomy one year before without relief from symptoms. Jaundice; bile in urine; fever; small gall bladder. Death ten days after operation from acute hæmorrhagic pancreatitis and fat necrosis.
18. 1912. Man, 65. *Cholecystostomy*.—Jaundice; ulceration of stones from common bile duct into pancreas. Death from acute pancreatitis on the day of operation.
19. 1912. Woman, 61. *Cholecystostomy and Choledochotomy*.—Jaundice; enlarged gall bladder. Death nine days after operation from hæmatemesis due to gastric ulcer.

It will be seen that fifty-four per cent. of the deaths are in this group: eight deaths from septic cholangitis and cholæmia, two deaths from pneumonia, two deaths from diffuse peritonitis, four deaths from hæmorrhage, two deaths from acute hæmorrhagic pancreatitis and one from hæmatemesis due to a chronic gastric ulcer. In two cases stones had been left in the common duct at a previous operation. In four, stones were found in the common and hepatic ducts post-mortem. The advanced nature of the cases is obvious and operation was done too late in the disease to save them.

### Conclusions

1. The mortality of cholecystostomy was the same as cholecystectomy. This was due to a careful selection of cases for cholecystectomy. Some of the cases which died after cholecystectomy would probably have been saved by cholecystostomy.

2. The mortality was due mainly to spread of the disease beyond the gall bladder before operation and the failure of the latter to relieve a desperate condition.

3. Cholecystectomy with choledochotomy is the ideal operation when stones are present in the common duct. The limitations of this operation are due to the complications present so frequently in these cases—jaundice, cholangitis, adhesions,

abscess round the gall bladder, fistulous communication with other hollow organs, pancreatitis.

4. Cholecystostomy with choledochotomy is the safer operation when stones are present in the common duct. The late results of this operation are good enough to make it unjustifiable to take the risk of routine removal of the gall bladder.

5. When stones are present in the common duct the gall bladder is usually inflamed, thickened, lying in a mass of adhesions and possibly surrounded by an abscess. From the point of view of treatment it must be compared with an acute appendicitis after the fourth day. It is unfair to say that such a gall bladder resembles the early stages of an acute appendicitis.

6. The slow progress of cholecystectomy was not due to conservatism but to the exigencies of the cases, which usually came under observation at a late stage of the disease.

7. A selection of cases with regard to age and extent of the disease will show a mortality for cholecystostomy or cholecystectomy as low as has been obtained in any part of the world.

8. The importance of a thorough exploration of the common ducts and the hepatic ducts, particularly when the gall bladder is contracted or surrounded by adhesions. This should be done with the scoop. The common duct should always be drained when stones have been removed from it. The question of cholecystectomy or cholecystostomy can then be decided on the exigencies of the case.

9. The risk of removing contracted, adherent gall bladders is greater than drainage and requires reflection and consideration.

10. When the common duct is infected, choledochotomy is the dominating factor because of the delay for two or more days of drainage of bile through the cholecystostomy tube. Drainage of bile into the duodenum may be facilitated by putting a rubber tube into the common duct with its lower end projecting into the duodenum.

11. The low mortality of cholecystostomy where the disease is confined to the gall bladder must be noted. Although the late results of cholecystectomy are better than cholecystostomy, the adherent gall bladder should not be removed as a routine.

12. That nine per cent. of the cases of cholecystostomy may require another operation does not justify the increased immediate risk of removing the gall bladder in every case.

13. In twenty per cent. of the fatal cases there was an abscess round the gall bladder. The value of preliminary drainage of the abscess must not be forgotten. This is a true parallel to the late appendix with adhesions. Where removal is dangerous, even a gangrenous gall bladder may be drained.

14. The mortality of second operations on the biliary apparatus is not so great as to justify the routine use of radical measures at the first operation.

15. In fifty-four per cent. of the fatal cases there was obstruction of and infection of the common bile duct with an abscess or adhesions round the gall bladder. Earlier operation before these complications is the most hopeful way of saving these lives.

## SARCOMA OF BONE

By R. P. ROWLANDS, M.S., Surgeon to Guy's Hospital.

SARCOMA of bone varies much in malignancy according to its structure and situation: when its essential cells are small and active it disseminates early, especially when it affects a bone or part of a bone which is rich in venous sinuses or is surrounded by very vascular soft parts. It is not surprising, therefore, that sarcoma of the femur or humerus is so malignant that it is almost certain to cause death within two years in spite of amputation or other treatment. This fact makes pathologist and surgeon ask whether amputation is worth while in these cases. In many instances it certainly is, for, although the chance of cure is small, the patient is saved much pain and misery from spontaneous fracture, hæmorrhage, fungation or secondary septic infection. These spontaneous fractures and wide local resections of growths of solitary bones are naturally followed by serious loss of function; moreover, local resection of sarcoma of these bones is nearly always followed quickly by local recurrence. On the other hand, sarcoma of some bones, such as the fibula, ulna or carpal bones, is far less malignant, and, as these or at least part of these bones are not indispensable, adequate local resection offers a good chance of relief or cure without disability or dissemination. These facts are illustrated by the cases related in this communication.

A study of the natural history of growths of bone teaches us that there are all degrees of malignancy, and that the same treatment is neither desirable nor applicable to all. It also teaches us to remove the growth whenever possible and to try every accessory method of treatment that appears to be hopeful. Of these deep x-ray therapy seems to be the most encouraging. I have not seen any improvement follow the use of Coley's fluid, although Coley and others in America warmly recommend its use in large doses.

### PERIOSTEAL SARCOMA OF LOWER HALF OF FIBULA; RESECTION; RECURRENCES; RADIOTHERAPY.

*Case 1.*—Lieut. H., R.N., was sent to me in 1913 for sarcoma of the lower half of the fibula (Fig. 1). There had been a

swelling there for some seven years, rapidly increasing of late. Amputation having been discussed and declined, the lower half of the fibula was widely resected with the invaluable aid of a tourniquet. It was not easy, however, to be certain that all the growth was removed. Some of the periosteum of the contiguous surface of the tibia and the projecting lips



FIG. 1.

Case 1.—Ossifying periosteal sarcoma of fibula. Excision of lower half of fibula in 1913.

of the inferior tibio-fibular joint were excised with a chisel. An external steel support with valgus strap enabled the patient to walk well, but it was discarded when war broke out to enable him to go on active service: he did so well that he was promoted to the rank of Lieutenant-Commander. It was wonderful to see him walk without the lower half of his fibula. There was no talipes valgus, the soft parts and scar tissue having consolidated so well as to support the ankle joint.

The recurrence shown in Fig. 2 was removed in 1922. The

growth had ossified and calcified in parts, but a section of the soft parts showed fibro-sarcoma. A streaky black shadow over the os calcis suggests the possibility of a recurrence there. In November 1923 the patient suffered so much pain and disability in the left foot, making it difficult for him to walk from his train to the Admiralty, that he sought treatment again. He was, by this time, anxious to have the leg amputated, but Dr. W. L. Watt agreed with me that deep x-ray



FIG. 2.

Case 1.—Local recurrence in 1922.

therapy should be tried. This was done by Dr. Watt, with excellent results up to the present (Figs. 3 and 4), the patient being free from pain and able to walk fairly well again. There are no signs of dissemination of the disease, which has almost certainly existed for twenty years.

In a somewhat similar case Bland Sutton<sup>1</sup> resected the upper half of the fibula for periosteal sarcoma, but the patient, having suffered from a local recurrence, died of dissemination of the growth in the lungs two and a half years later.

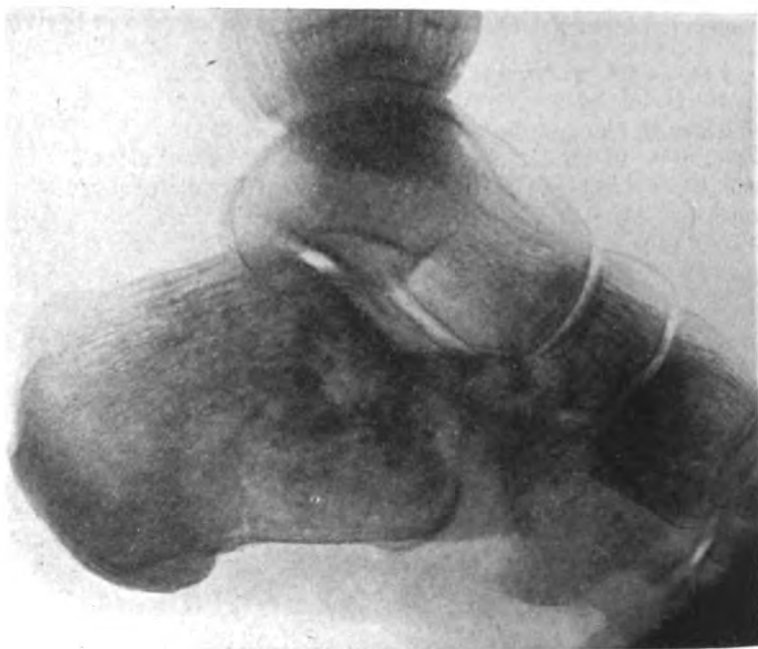


FIG. 3.

Case 1.—Showing widespread involvement of os calcis, tarsal bones and interosseous joints.

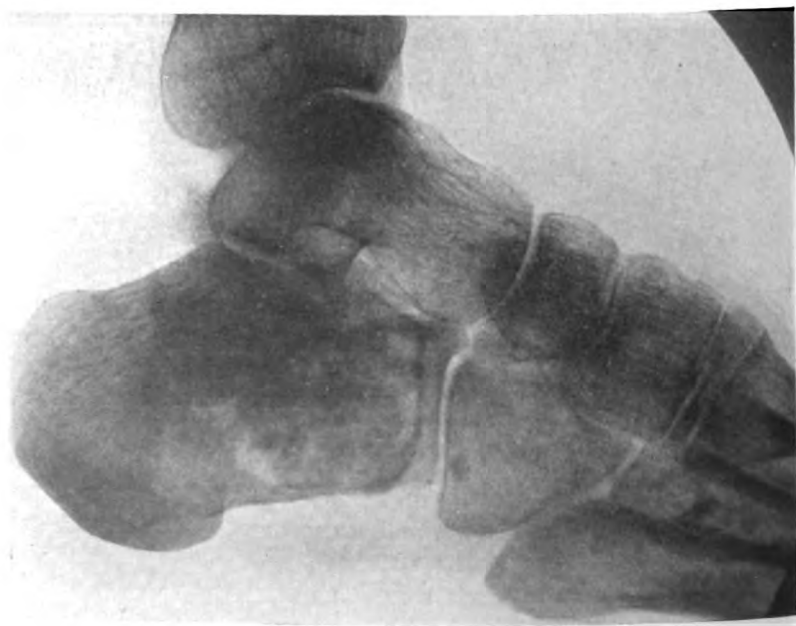


FIG. 4.

Case 1.—Showing new bone formation with striation and normal tarsal bones and interosseous joints, and that the fourth metatarsal had also been involved as well as the os calcis and tarsal bones.

## CENTRAL SARCOMA OF RADIUS; RESECTION WITH GRAFTING.

*Case 2.*—Miss X. J., aged eighteen, sustained in 1915 a greenstick fracture of the left radius from a very slight injury and was treated by a distinguished surgeon in Edinburgh. There was some delay in securing union. The reason for this is shown in Fig. 5, although neither the surgeon nor the radiographer seems to have noticed it. The pale area in the centre of the radius at the site of the fracture indicates an early growth,

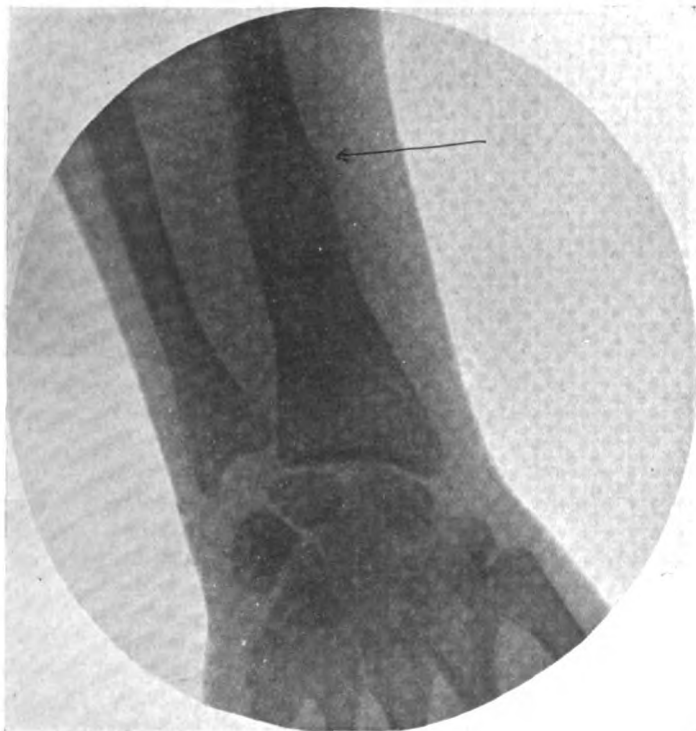


FIG. 5.

Case 2.—Showing early central growth at seat of greenstick fracture in 1915.

which was afterwards proved to be a fibro-sarcoma. In 1919 the same bone was broken by a slight fall while roller skating, and the patient was admitted into a London hospital, where a radiogram was taken. Again, neither the surgeon nor the radiographer noticed any suspicious abnormality in addition to the fracture. A few days later Mr. Coldwell took a radiogram (Fig. 6) which conclusively confirmed the suspicion of a central growth. At first this was thought to be a myeloma, although it was situated in the shaft and not in the epiphysis, which is the usual site of myeloma. Exploration of the growth



from behind and with the aid of a tourniquet showed a fibrous growth much firmer than a myeloma. This was completely removed, as was proved later, without refracturing the bone, the anterior and internal walls of the cavity being left entire and healthy after careful scraping with a sharp spoon. The section, which was examined by Dr. G. W. Nicholson, Professor Shattock and others, showed ossifying fibro-sarcoma, which

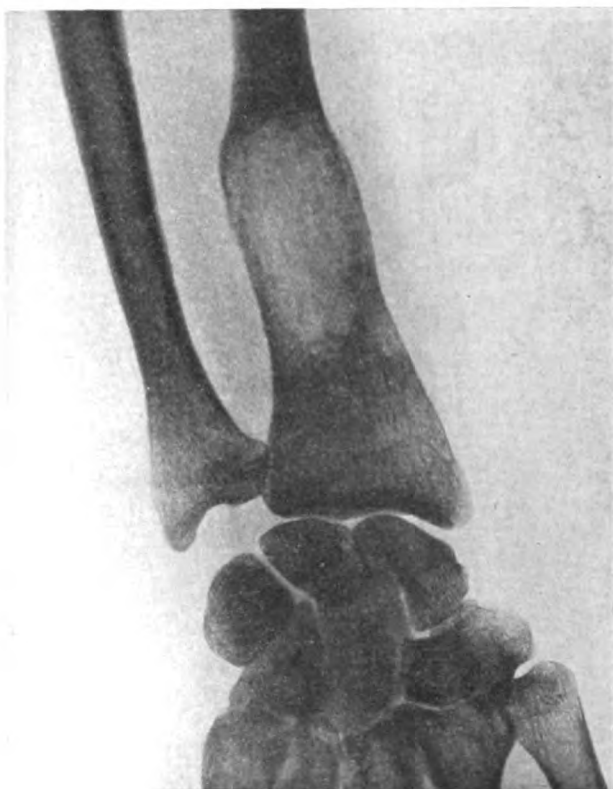


FIG. 6.

Case 2.—Showing increase of central growth in 1919 when re-fracture had taken place.

was thought to be of a low grade of malignancy. Four surgeons were consulted and various suggestions were made by the pathologists and surgeons as to treatment: these included amputation of the forearm, excision of the whole radius, and resection of a third of the radius together with a corresponding length of ulna. Ultimately, in April 1919, I resected  $2\frac{1}{2}$  inches of radius and immediately inserted in the gap a piece of the middle of the fibula nearly three inches long. The wound healed well, the graft united, and the patient used the forearm and hand very well with and without the aid of a sup-

porting instrument made by Mr. F. G. Ernst. Ultimately the union at the upper end of the graft gave way after a slight injury, and the graft began to wither from above downwards (Fig. 7). A long plate was inserted in 1920 and restored the waning strength of the limb. At the same time a piece of the posterior part of the shaft of the radius was shifted down to bridge the gap between the refreshed surfaces at and near the



FIG. 7.

Case 2.—Showing fracture at the upper end of graft in 1920.

upper end of the graft, and before long reunion took place (Figs. 8 and 9). The patient still wears the plate without trouble, but the graft refractured in 1922 near its middle, when the patient was steering a Ford car over a rough road in Africa. This fracture has united, but the hand has become a little abducted owing to shortening of the graft. The patient uses the hand fairly well with the aid of a block leather support.

There has been no sign of recurrence of the growth in the ten years since it appeared and six years since it was removed. A longer graft overlapping the shaft of the radius would have given a better result in this case, but driving the lower end of the graft into the cancellous lower end of the radius was very satisfactory in that it caused rapid and permanent union.



FIG. 8.

Case 2.—Showing plate, with imperfect union of graft, in Feb. 1921.

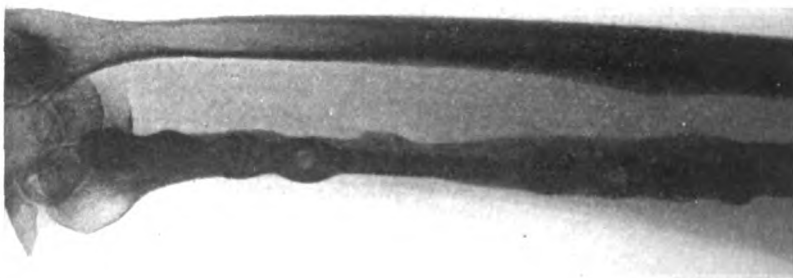


FIG. 9.

Case 2.—Showing union taking place in August 1921.

#### SARCOMA OF RIBS; RESECTION.

*Case 3.*—Mrs. B., aged 48, was sent to me for operation in February 1921. A swelling had appeared over the lower right ribs some fifteen months before. It was then thought to be a lipoma and the patient did not appear again for more than a year, when the swelling had greatly enlarged and become fixed. When I saw her the growth was five inches long and four inches broad and inseparable from the lower ribs.

Ethyl chloride, warm ether and oxygen were administered through an ordinary facial mask, and a large elliptical mass of the whole thickness of the chest wall (including the tumour)

was excised, thus making a very large opening into the right pleura. There was little, if any, shock. The whole of the right lung, which had partially collapsed, the dome of the diaphragm, the wall of the pericardium and the descending aorta were seen and examined. All bleeding having been arrested and the pleural cavity cleared of clots, the thoracic wall was closed in layers, pleura to pleura, muscle to muscle, and skin to skin. This was easier than was expected, for there was little tension upon the stitches. The patient did well, the wound healing primarily and the pneumothorax rapidly absorbing: there was no pleurisy or fever. The patient was able to travel home ten days later and has remained well ever since. The growth was a slow-growing, spindle-celled sarcoma.

Such comparatively favourable cases of sarcoma of bone are not uncommon, and I believe that their occurrence accounts for some of the cures credited to special methods of treatment, such as Coley's fluid, radiation and so on. When the natural history of the disease varies so much, it is necessary to take a broad view in judging the efficacy of any method of treatment.

Some twenty-five years ago I removed a central ossifying fibro-sarcoma from near the symphysis of the lower jaw of a nurse at Guy's Hospital. It had displaced, destroyed or loosened the roots of five teeth. The growth was shelled out, and the walls of the cavity were thoroughly scraped. No recurrence has taken place.

About eight years ago I excised the inner third of the clavicle, containing a central sarcoma (not a myeloma), from a young girl. There was no recurrence during the five years we were able to keep in touch with her, but she cannot now be traced, having moved away from the district.

Fourteen years ago I removed the left fore-quarter of a young woman for extensive periosteal fibro-sarcoma of the scapula. The operation had been refused at another hospital owing to the extent of the growth. Nine years later a recurrent nodule appeared and was removed from the second rib.

Mr. L. A. Dunn removed the scapula of a young woman in 1906, in Guy's Hospital, for periosteal sarcoma, and there was no sign of recurrence nineteen years later. The functions of the limb were good, except for abduction of the arm, which was very limited.

#### DIAGNOSIS

The diagnosis of sarcoma of bone is greatly facilitated by x-ray examination, and experienced radiographers and surgeons can generally recognise the appearances characteristic of this

disease, whether periosteal or central, but in many cases it is still necessary to explore and take a section before undertaking any radical treatment, especially amputation. Exploration, especially with the aid of a tourniquet to limit the risk of embolism of growth, can do no harm and may be a limb-saving measure.

*Central cysts.*—Amputation at the right shoulder joint had been advised in the case of a boy (aged 8 years) as the result of clinical and radiographic examinations indicating a sarcoma near the upper end of the humerus. The parents declined amputation without exploration and changed their surgeon. Subsequent exploration showed the swelling and spontaneous fracture present to be due to a central simple cyst containing cholesterol crystals. The boy made a complete recovery with full use of his arm and is now well, some eight years later.

*Fibro-cystic disease.*—This is very liable to be mistaken for sarcoma. Mr. W. H. Ogilvie operated on a small boy for me at Guy's Hospital, who was supposed to have a central sarcoma of the shaft of the humerus. Exploration and microscopic section, however, proved that fibro-cystic disease was present, and resection of the greater part of the shaft of the humerus was fortunately followed by complete recovery of the function of the limb. Insertion of an autogenous graft proved to be unnecessary, for blood filled the gap and ossification took place without the aid of a graft.

*Myeloma.*—Many amputations have been needlessly performed for myeloma of bone before the danger of mistaking it for sarcoma containing giant cells was recognised. It is therefore of the greatest importance to explore and take a section of all central growths at the epiphysial ends of the long bones. It is also wise to submit the section to several pathologists.

*Osteoma.*—I had a case recently in which an osteoma of a metacarpal bone was mistaken for sarcoma by a surgeon and pathologist and amputation suggested. This, however, was avoided after further examination of the section by two pathologists and consultation with two surgeons.

*Chondroma.*—A gamekeeper came to me in 1916 with a large growth, supposed to be sarcoma, in his right leg, but radiographic examination by Dr. W. Lindsay Locke proved the tumour to be a calcifying chondroma, showing the dense black spots characteristic of this condition. Local removal of the growth, which was growing from the tibia, proved very satisfactory, the man being well and able to do his work nine years later. The x-ray findings were confirmed by multiple

microscopic sections, without which it is easy to overlook the sarcomatous tissue in a chondrifying sarcoma.

*Periosteal lipoma.*—Some years ago a woman was sent to me at Guy's Hospital to have the arm amputated for periosteal sarcoma of the upper end of the radius. Radiographic examination showed ossification of the deeper parts of the growth, but upon exploration the latter proved to be a periosteal lipoma with ossification of the deeper parts of its capsule, so that local removal gave a very satisfactory result.

*Fracture with non-union.*—There is in the Guy's Museum a forearm which was amputated elsewhere many years ago under the impression that a sarcoma had caused spontaneous fractures of the two bones of the forearm. Subsequent examination of the specimen showed non-union of fractures, the history of which had been concealed by the patient. Exploration might have prevented this disastrous error.

*Osteitis.*—Without blood examination and exploration of the swelling it is easy to mistake osteitis, especially syphilitic osteitis, for sarcoma. Central abscess has also been mistaken for sarcoma.

*Hydatid.*—Central hydatid disease of the bone has been mistaken for sarcoma, but this error should be avoided by blood examination and exploration.

#### TREATMENT

Sarcoma of bone calls for early and wide resection before dissemination and serious local destruction takes place. The orthodox treatment of sarcoma of the long bones is amputation well above the disease, using only skin flaps. For instance, sarcoma of the femur has been treated by amputation at the hip joint, leaving only skin flaps. It is unfortunate, however, that high amputation in the large majority of cases fails to prevent dissemination, which has, in fact, often taken place before the patient comes for treatment, so that amputation is like shutting the stable door after the horse has been stolen. It is therefore of the greatest importance to examine for secondary growths by all available methods, especially radiography, before advising an amputation that may not only be useless but vexatious and prematurely disabling. Further, it is always worth while considering local resection, granted that the function of the limb can be preserved without adding to the risk of general dissemination or local recurrence. When amputation of the thigh has to be performed for sarcoma of the lower part of the femur with threatening or actual spontaneous fracture, it is worth while considering whether ampu-

tation at the hip joint, a most disabling operation, is really better than amputation through the upper third of the thigh, which leaves a useful stump for the patient for the remainder of his life, whether this be long or very short. Again, extensive local resection with grafting, following radiation and other accessory methods of treatment, is also worth considering, seeing that the life-saving results of amputation are so negligible. When dealing with twin bones, such as the tibia and fibula, and especially with the important bones of the forearm and hand, local resection is more hopeful both as regards the avoidance of local and general recurrence and the preservation of function of the limb.

#### CONCLUSION

In conclusion, it appears that every case of sarcoma of bone requires special consideration and treatment. The outlook is not so gloomy as is generally supposed, early and wide removal of the growth aided by the natural resistance of the body and accessory methods of treatment being not uncommonly followed by good results or even permanent recovery.

#### REFERENCE

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## AN ANALYSIS OF THE RESULTS IN FIFTY CASES OF TRANS-SEPTAL ORCHIDOPEXY FOR IMPERFECTLY DESCENDED TESTICLE

By PHILIP TURNER, M.S., Surgeon to Guy's Hospital.

SINCE 1912, with the exception of a few cases in adults where excision has been necessary, I have treated all cases of imperfectly descended testicle by bringing the malposed testis through the septum into a bed which has been prepared for it in the opposite side of the scrotum. Detailed accounts of the operation have been given elsewhere (<sup>1</sup>, <sup>2</sup>), but the chief stages may be briefly recapitulated.

(1) A small incision is made in the external oblique just above the internal abdominal ring. The internal oblique is drawn upwards and the spermatic cord, enclosed with the patent funicular process in a sheath derived from the infundibuliform fascia, is exposed just below the internal ring.

(2) The spermatic cord and the testicle are drawn out from the inguinal canal into the wound: the hernial sac, or the patent processus vaginalis, which is nearly always present, is separated from the vas and the veins after the fascial sheath has been opened, and is ligatured above at the internal ring and below just above the tunica vaginalis.

(3) The fascial sheath of the cord is divided, and the attachment of the gubernaculum to the tissues of the groin is torn through.

(4) The gubernaculum, just below the testicle, is transfixated and ligatured: the ends of the ligature are left long and are secured by a pair of Spencer Wells forceps; the redundant portion of gubernaculum beyond the ligature is cut away.

(5) An incision is made on the opposite side of the scrotum and a bed for the testicle to be transplanted is made between the septum and the normally placed testicle.

(6) The forceps with the ends of the ligature are introduced through the incision in the external oblique and are directed along the inguinal canal, through the external ring, and well down into the scrotum; they are then made to impinge against the septum, which is pushed before them into the scrotal wound.

(7) A small incision is made in the septum over the points of the forceps, which are then pushed through and the ends of the ligature are secured.



(8) The forceps are withdrawn slowly and are opened from time to time so as to make a way for the passage of the testicle, which is drawn into its new position by traction on the ligatures.

No sutures are required to fix the testicle in its new position; retraction is prevented by the elastic contraction of the small opening in the septum.

In bilateral cases an interval of about six months is allowed between the two operations in order to ascertain that the first operation has been successful before the second is undertaken.

In November 1914 I described and showed two cases, one bilateral and one unilateral, six months after operation, at the Section for the Study of Disease in Children of the Royal Society of Medicine.<sup>3</sup> The results in both children were very satisfactory.

In 1919 I attempted to collect all the cases, twelve in number, on whom I had operated in 1914. Unfortunately I was only able to trace three cases, two children and one adult (Nos. 1, 2, 3).<sup>\*</sup> The former, one of whom had been operated upon for a bilateral deformity, I showed at the Section for the Study of Disease in Children in March 1919, about 4½ years after the operation.<sup>4</sup> The results were most satisfactory. On examination, though the hernia scars were visible, both patients appeared to be perfectly well-developed boys with normal testicles, and even on close examination there was nothing to suggest that any transplantation of the testicles had ever been carried out. The result in the adult patient was equally satisfactory (Case 3). The two patients shown in 1914 could not be traced.

Since the war I have, with the assistance of my House Surgeon, arranged for all patients living in London or near suburbs, who were operated upon in any year, to attend on the same afternoon about the end of the next year for examination and comparison of results. The examination was thus, with the exception of the 1919 cases, where the patients were collected as early as March 1920, made somewhere between one and two years after the operation. The results thus noted at the time are shown in the table below. Most of the patients have, of course, been seen on other occasions, at Out-patients or in the wards, but in no case has there been any need to add to or alter the result recorded at the systematic inspection.

In collecting a series of results it is desirable that the preliminary diagnosis, the treatment, the after-treatment, and the recording of results should be carried out in all the cases according to the same plan. Thus all the cases in the present series

\* These numbers refer to the list of cases on p. 218.

were operated upon in the general wards: the diagnosis—not always an easy matter, as I have pointed out elsewhere<sup>5</sup>—was made at Out-patients and amply confirmed by subsequent examinations in the ward; and at the investigation of results I have had the assistance of House Surgeon and dressers, who have previously collected the reports with notes of the condition on admission and of any special points noticed during the operation.

No private patients, and no patients who live at such a distance from London as to render it a difficult matter for them to come up for inspection, are included in the present series. I have, however, seen or heard from many of these from time to time, and the results obtained in these cases are much the same as in the list of hospital patients given below. Of the patients requested to attend for examination about four-fifths presented themselves—a very satisfactory proportion.

From observation of a considerable number of cases I have come to the conclusion that the condition found six months after the operation enables one to judge as to whether the operation is a success or a failure. That is, if the transplanted testicle is in its normal position in the scrotum, if there is no thickening or adhesion around it, and if it has increased in size and is of normal consistence, the patient will continue to do well. On the other hand, if the testicle is going to atrophy, this will be quite obvious at the end of six months, and no further improvement can then be expected.

The results recorded are essentially anatomical results. It is generally recognised that, though the imperfectly descended testicle usually produces its internal secretion so that the secondary male characteristics are normally developed, the important function of spermatogenesis is, as a rule, absent, so that when the deformity is bilateral the patient will probably be sterile. In the present series of cases it is impossible to speak as to the physiological result, as this can only be done by tracing the history of patients with a bilateral deformity who have reached adult life. None of the bilateral cases in the present list are yet of an age to render this possible. It may, however, be stated that in the great majority the anatomical result has been so satisfactory as to lead one to hope that if the deformity is rectified before the final developmental stages which take place at puberty, there is, at any rate in a good proportion of cases, a prospect that this important function may be normally carried out. Thus the operation should, whenever possible, be performed before puberty, but Case No. 3 shows that it is possible to obtain an excellent anatomical result even in an adult.

The following are the points to which attention was directed in the investigation of the results :

(1) Post-operative complications. These are infrequent and of no great importance. Suppuration of the scrotal wound occurred after two operations, both on the same patient for a bilateral condition. The patient was a boy aged 6, Case No. 23, of poor mental development, who was found, after the first operation, interfering with his dressings. In spite of the infection the result of the first operation was excellent. On the second occasion there was a hæmatoma of the scrotum which became infected; the testicle was poorly developed and it subsequently atrophied. One other case in this series developed a hæmatoma of the scrotum, but here the blood was absorbed and the result was perfectly satisfactory. One patient, Case No. 10, had a small hydrocele of the cord, presumably from a small portion of the funicular process which had been left. This cleared up with palliative treatment, as did also a case not in this series who developed a small hydrocele of the tunica vaginalis. I also know of another patient, not in the present series, who has developed a varicocele.

It is important to note that there is no particular pain after the operation, certainly no more than after an operation for hernia.

(2) Hernia. In 17 of the 43 patients the presence of a hernia was definitely diagnosed before the operation. In the majority of the remaining cases, though no actual hernia was present, a patent funicular process, or potential hernia, was present, and the sac was then removed at the operation. In three cases only, Nos. 25, 35, 36, was it definitely noted in the report at the time of operation that no hernial sac or patent funicular process could be found. Unfortunately this negative point is apt to be overlooked in writing the account of the operation, and hence no inference from these figures as to the frequency of this occurrence is possible. My impression is that no hernial sac or patent funicular process can be found in somewhere between 5 and 10 per cent. of all cases.

It is interesting to note that in every case the hernial scar was perfectly sound and that there was no sign of recurrence. This is in accordance with the excellent results usually obtained after operations for hernia in children.

(3) The size and consistence of the testicle. The imperfectly descended testicle is usually distinctly small and poorly developed when compared with the normally placed organ in unilateral cases. Hence in the investigation of results close attention has always been directed to the size and consistence of the testicle. In the great majority, all those in which the

result is described as "Class A," there has been no appreciable difference in size between the transplanted and the normally placed testicle. Indeed in certain cases (Nos. 1, 13, 14, 15, 20, 35, 39) the testicle which has been brought down into the scrotum has been distinctly the larger. On these grounds it is claimed that transplantation of the testicle, at any rate before puberty, is followed by growth in size and possibly by normal physiological development.

In bilateral cases, where there is no normal testicle for comparison, it is necessary to contrast the transplanted organs with those of a normal boy of the same age. In these cases, however, the increase in size is often more striking than in the unilateral cases, and in patients where the operation on both sides has been successful (Nos. 2, 11, 27, 31), the patient appears to be a perfectly normal boy.

The consistence of the testicle is also of importance. The successfully transplanted testis is firm and elastic and cannot be distinguished in this respect from the normal organ. The testicle which has atrophied is, on the other hand, small, soft and flabby; or it may have practically disappeared, remaining only as a small fibroid nodule. A small testicle which has not atrophied feels very different from one in which atrophy has definitely occurred (Nos. 21, 24, 43). In no case in the series was the testicle adherent to the scrotal scar, neither was there any thickening or induration around the testicle or along the spermatic cord. This is a point of some importance, for, after the operation, there is always some oozing of blood into the bed made for the testis in the cellular tissue of the scrotum, as well as into the track opened up by the forceps for its passage from the inguinal canal into its new position. The result is that if the wound is examined a week or two after the operation, an irregular shapeless mass will probably be felt in the scrotum, and hard, rigid thickening along the course of the cord. This, of course, is due to blood which has collected and clotted around the testicle and cord. It all becomes absorbed in about four to six weeks and completely disappears. There was no trace of any induration or thickening around the testicle or cord in any of these 50 collected cases. A collection of blood worthy of being called a hæmatoma is unusual, but even when it does occur will probably be absorbed.

Atrophy of the testicle occurred in 7 cases only (14 per cent.) in the present series. In several of these the condition found at the operation offered a very poor prospect of success, and notes of this were made in the report at the time (Nos. 16, 23, 30, 41). On the other hand, in certain cases where the

testicle was small and poorly developed and a good result was not anticipated the patient did remarkably well (Nos. 12, 32). For this reason I have performed the same operation in all children and young adults whether the condition found was favourable or not.

(4) The position of the testicle. After transplantation no sutures are used, but the testicle is retained in its new position by the contraction of the small opening in the septum through which it has been made to pass. The result is that there is a continuous slight force acting over an indefinite time, counteracting any tendency to retraction. If sutures are employed to fix the testis in the scrotum, these absorb or cut through after a few days and their action then ceases.

With the exception of cases where the vas or the spermatic veins are unduly short there is no doubt that the testicle remains in its new bed and shows no tendency to retract. In my experience undue shortness of the vas or the veins is very unusual if the operation is performed before puberty. It is much more common in patients over the age of eighteen years, and in these, if the testicle cannot be brought down into the scrotum and the other testicle is normal, excision is indicated. Case No. 3, however, shows that a very satisfactory result may sometimes be obtained in an adult when the vas and veins are of sufficient length.

In 70 per cent. of the present series, all those where the result is Class A, the transplanted testicle is well down in the scrotum with no appreciable difference in this respect from the normally placed testis of the opposite side. Indeed not infrequently the testicle which has been brought down is distinctly lower than the other (Nos. 8, 9, 10, 36). This depends upon the fact that in the process of freeing the testicle during the operation the cremaster is torn through when the fascial sheath of the cord is divided. For this reason on examination of unilateral cases it is commonly found that the cremasteric reflex is absent on the side which has been operated upon: in bilateral cases it is absent on both sides. The condition of the cremasteric reflexes is mentioned in a certain number of the cases in the list given below, but though it is not mentioned in others, absence of the cremasteric reflex was noted on the side of the operation in practically all the cases.

When the spermatic cord is examined in successful cases it also appears to be perfectly normal. There is no thickening or induration, and no feeling of tenseness or tautness. Indeed the testicle is suspended in the scrotum in a perfectly natural manner. Immediately after the operation the cord often feels

as though it were slightly on the stretch. It is thus impossible to avoid coming to the conclusion that not only does the testicle increase in size, but that also growth in length occurs in the vas and other structures of the spermatic cord.

(5) The condition of the scrotum. The scrotum quickly adjusts itself to its contents. If a testicle be removed, that side of the scrotum soon shrinks and contracts. In a patient with a hydrocele or a scrotal hernia the scrotum enlarges in accordance with the size of these abnormal contents. Thus if the two testicles are in the same side, that half of the scrotum enlarges while the opposite empty side contracts. In bilateral cases of imperfect descent of the testicle the scrotum is often represented by a corrugated patch of skin. After orchidopexy has been performed, even in these cases, the scrotum will enlarge and eventually present a normal appearance (Case No. 2). The scrotal scar practically disappears, and in most cases can only be detected with difficulty on close inspection.

(6) Pain or other disability. In no case has the patient complained of any pain or any other disability. The transplanted testicle is not liable to attacks of inflammation. In no case has excision or any secondary operation been required.

(7) General condition. In several cases the parents have volunteered the information that the intelligence and mental condition have improved. No special note has been made of this, as verification is impossible and it may be simply the normal growth and development of the child.

In order to avoid numerous repetitions of the same conditions the results have been classified in three groups, A, B and C, one of which letters is found in the result column opposite each operation. The condition found in each of these groups is as follows :

*Group A.*—The testicle hangs normally and easily, well down in the scrotum, at appreciably the same level as the normal testis. The testicle is approximately the same size and consistence as the normal one; it is not adherent to the scrotal scar and there is no induration around it or the cord. The scrotal scar is invisible or can only be detected on close examination. There is no pain and no disability. The hernia scar is normal. Cases in which the result is described as "A" are thus required to reach a very high standard, which may be summarised by saying that on examination the patient appears to be a normal boy who has been operated upon for hernia, and where, unless one knew the history of the case, nothing would be noticed to suggest that the patient had ever been operated upon for imperfectly descended testicle.

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Of the 50 operations the result in 35, or 70 per cent., belong to this group.

*Group B.*—The testicle shows some abnormality. It is of normal consistency, but though it is not atrophied it is either distinctly smaller than the normal testicle or is situated at a higher level, in the upper part of the scrotum: usually it is both smaller and higher. There is no pain or other disability.

Though on examination an obvious abnormality of the testicle is found, the "B" results are by no means to be regarded as failures. Indeed some striking successes are to be found in this group. The following two are examples:

Case No. 32. Here the right testicle could not be felt at all before the operation. It was found in the abdomen and only drawn down with difficulty. A year afterwards the testicle was well down in the scrotum but was distinctly smaller than its fellow: it was of normal consistence and not atrophied.

Case No. 12. This patient had been operated upon three years before at another hospital, when a hernial sac was removed and the testicle pushed back into the abdominal cavity. It, however, remained at the upper part of the inguinal canal and was the cause of chronic pain and disability. At the second operation the imperfectly descended testicle was brought down from the inguinal canal to the opposite side of the scrotum, with the result that pain and disability completely disappeared. Having undergone such vicissitudes it is not surprising to find a year later that "it is slightly higher and smaller than the left (normal) testicle."

*Group C.*—Cases where the result is described as "C" are failures. The testicle is small, soft and atrophied: in some cases it has practically disappeared. There is no pain and no disability. In some of these cases there was, at the operation, some abnormality of the testis which made one feel that a successful result was very improbable, but in others the testicle appeared to be in good condition, the vas and veins of good length, so that there was every reason to hope for a successful result. The imperfectly descended testicle, the vas and the veins are doubtless always delicate structures, and one can only suppose in these cases that, possibly as the result of manipulation, some injury was inflicted leading to bruising of the testicle or vas, or possibly thrombosis of the vessels. It is on this account that in bilateral cases an interval of some months is allowed to elapse between the two operations, so that the result of the first operation is seen before the second is undertaken.

In the following list of cases there are in all 43 patients. Of these, 7 were bilateral cases in which the operation was

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performed on both sides, 6 were bilateral cases in which only one side has been operated upon, while 30 were unilateral cases. The total number of operations is thus 50. Of these the result in 35, or 70 per cent., is extremely satisfactory and belong to Class A. In 8, or 16 per cent., the result, though good, is not in all respects satisfactory, Class B. In 7, or 14 per cent., the testicle has atrophied and the result is a failure, Class C.

It is difficult to make a comparison with the results obtained with other methods of orchidopexy, as I am not aware of any published series of cases.

McAdam Eccles<sup>6</sup> thus sums up the results obtained after the older method of orchidopexy: "In a certain proportion of cases it will grow and develop so as to become in the future a thoroughly efficient organ. The exact number of instances in which this happy termination does occur is unknown, because there are no proper statistics on the subject. But, from the after-inspection of not a few cases where the testis has been transplanted into the scrotum at the same time as a radical operation has been performed upon the accompanying inguinal hernia, it has been found that the testis has a very great tendency to mount again into the region of the superficial ring, if not actually into the inguinal canal itself, particularly if the case is observed some years after the initial operation. With regard to the second half of the question (that of the possibility of further growth and development), if the testis does remain in position it will in some instances undergo development; but, on the other hand, should it be retracted, it will as certainly become more atrophied, or, at any rate, does not develop. There is here, once more, a significant want of definite record of the subsequent history of these cases, and those that have been traced have not given the operator too much satisfaction."

I have not employed the older method of suturing the testicle into a bed prepared for it in the scrotum on the same side, for many years, but I am quite in agreement with these remarks. Using the present standard of results, I am sure that the great majority of my old cases would belong to Class C, with perhaps an occasional B. I cannot recall ever having obtained a result which I would include in Class A.

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- <sup>3</sup> *Proc. Roy. Soc. Med.*, Sec. for Study of Disease in Children, viii. 17, 1914.
- <sup>4</sup> *Proc. Roy. Soc. Med.*, Sec. for Study of Disease in Children, xii. 60, 1919.
- <sup>5</sup> P. Turner: *Guy's Hosp. Rep.* lxxii. 328, 1922.
- <sup>6</sup> W. McAdam Eccles: *The Imperfectly Descended Testicle*, p. 39.



No.	Name.	Age in years at date of operation.	Diagnosis.	Date of operation.	When seen.	Result.	Remarks.
1	Peter F.	5	Lt. I. D. T. & Lt. Ing. H.	Dec. 1914.	March 1919.	A.	Left testis is slightly larger than the right.
2	David E.	9	Bilateral I. D. T. & Ing. H.	Left side, July 1914. Right side, Dec. 1914.	March 1919.	A. } A. }	Testes normal in size and consistence. Both have increased in size and the scrotum has developed.
3	George O.	39	Lt. Ing. H., ? strangulated Lt. I. D. T.	Aug. 1914.	March 1919.	A.	Admitted for ? strangulated Ing. H. Left testicle fairly well developed and so was transplanted. Both testicles small but approximately equal in size. Patient a married man, five children.
4	Albert A.	16	Rt. Ing. H. & Rt. I. D. T.	March 1919.	29/3/20	B.	Right testicle slightly smaller and rather higher than left.
5	David W.	21	Lt. Ing. H. & Lt. I. D. T.	June 1919.	29/3/20	B.	Left testicle slightly higher than right.
6	Alfred S.	14	Lt. Ing. H. & Lt. I. D. T.	April 1919.	29/3/20	C.	Testicle small, soft and probably atrophied.
7	Walter T.	12	Double I. D. T.	Oct. 1919.	29/3/20	A.	Left side operated on. Right side had been operated on at another hospital, but testis had retracted into inguinal canal.
8	Thomas S.	14	Rt. Ing. H. & Rt. I. D. T.	Oct. 1919.	29/3/20	A.	Right testicle slightly lower than left.
9	Wm. S.	13	Lt. Ing. H. & Lt. I. D. T.	April 1919.	28/3/20	A.	Left testicle slightly lower than right.
10	William P.	11	Lt. Ing. H. & Lt. I. D. T.	Sept. 1919.	29/3/20	A.	Tiny hydrocele of cord, which eventually cleared up. Left testicle slightly lower than right.
11	Joseph A.	11	Bilateral I. D. T.	Right side, Oct. 1920. Left side, Aug. 1921.	8/1/23 9/1/23	A. } A. }	Both testicles increased in size. Cremasteric reflexes absent.

# IMPERFECTLY DESCENDED TESTICLE 219

No.	Name.	Age in years at date of operation.	Diagnosis.	Date of operation.	When seen.	Result.	Remarks.
12	Harold B.	18	Rt. I. D. T.	Feb. 1920.	14/11/21	B.	Previous operation in 1917, when testis was pushed back into the abdomen and a hernial sac removed. Pain persisted after this. Right testicle slightly smaller and higher than left.
13	Arthur G.	8	Lt. I. D. T.	Dec. 1920.	14/11/21	A.	Left testicle slightly larger than right. No left cremasteric reflex.
14	Fred C.	9	Lt. I. D. T. & Lt. Ing. H.	Nov. 1920.	14/11/21	A.	Left testicle slightly larger than right. No cremasteric reflex.
15	Sam W.	5	Lt. I. D. T. & Lt. Ing. H.	Dec. 1920.	14/11/21	A.	Left testicle slightly larger than right. Left cremasteric reflex absent.
16	Richard C.	9	Rt. I. D. T.	Dec. 1920.	14/11/21	C.	Testicle atrophied. Note in report: "The testicle was atrophied and it was doubtful whether it should not be excised."
17	Albert H.	18	Rt. I. D. T.	July 1920.	8/1/22	A.	Testicle well developed; same size as the left. Right cremasteric reflex absent.
18	Alfred K.	13	Rt. I. D. T.	March 1921.	6/1/23	A.	Right testicle a trifle smaller than left.
19	Walter F.	16	Rt. I. D. T. & Rt. Ing. H.	Feb. 1921.	6/1/23	A.	Occupation a blacksmith. No disability.
20	John S.	14	Lt. I. D. T.	March 1921.	6/1/23	A.	The transplanted testicle is slightly larger than the right.
21	Horace S.	9	Bilateral I. D. T.	March 1921.	6/1/23	B.	Right testicle small but not atrophied and of good consistence. It is situated in the upper part of the scrotum. Left testicle was represented by a small fibroid nodule in the scrotum. No operation was done on this side. Noted in the report that the vas was very short.
22	Eric L.	7	Lt. I. D. T.	Nov. 1921.	6/1/23	A.	Admitted with the diagnosis of bilateral I. D. T., but it was found that the right testicle could be manipulated into the scrotum. An example of "Spastic Retraction." Note in Report that at the operation the testicle was found to be small and poorly developed.

No.	Name.	Age in years at date of operation.	Diagnosis.	Date of operation.	When seen.	Result.	Remarks.
23	Herbert D.	8	Bilateral I. D. T.	Right side, Oct. 1921. Left side, March 1922.	6/1/23	A. C.	Note in report that at the second operation the left testicle was small and atrophied and that a good result was unlikely. Suppuration after each operation.
24	Benjamin W.	8	Bilateral I. D. T.	Left side, Dec. 1920. Right side, April 1921.	6/1/23	A. B.	Right testicle is slightly smaller than the left but is not atrophied. Noted in the report that the right testicle was small and that the vas was short.
25	Lionel J.	9	Rt. I. D. T.	July 1921.	6/1/23	A.	No hernial sac found. Testicles equal in size.
26	Fred M.	21	Rt. I. D. T. & Rt. Ing. H.	May 1921.	6/1/23	B.	Right testicle smaller than left and situated at a higher level. Not atrophied. Noted that right testicle was small and poorly developed at the time of the operation.
27	George B.	6	Bilateral I. D. T.	Right side, June 1922. Left side, Dec. 1922.	14/1/24	A. A.	Both testicles have increased in size.
28	Herbert McC.	11	Lt. I. D. T.	April 1922.	14/1/24	C.	Testicle has atrophied and practically disappeared.
29	Richard H.	6	Rt. I. D. T.	Feb. 1922.	14/1/24	A.	Testicles small but equal in size. Left testicle not completely descended but does not require operation. Hematoma after the operation.
30	John C.	6	Bilateral I. D. T. & Rt. Ing. H.	July 1922.	14/1/24	C.	Testicle atrophied. Note from report: "A small testicle could be demonstrated at the operation."
31	Charles A.		Bilateral I. D. T.	Right side, May 1922. Left side, Dec. 1922.	14/1/24	A. A.	No cremasteric reflex either side. Both testicles well developed.
32	Albert P.	16	Rt. I. D. T. & Rt. Ing. H.	Jan. 1922.	14/1/24	B.	Testicle though well developed is distinctly smaller than the left. Operation note: "The right testicle was in the abdomen and was only drawn down with difficulty."

# IMPERFECTLY DESCENDED TESTICLE 221

No.	Name.	Age in years at date of operation.	Diagnosis.	Date of operation.	When seen.	Result.	Remarks.
33	Leonard D.	10	Lt. I. D. T. & Lt. Ing. H.	March 1923.	15/12/24	C.	Testicle atrophied. Could be felt as a small fibrous nodule. Note in report: "Testis and vas found to be well developed, but their vascular supply was poor."
34	George W.	17	Rt. I. D. T.	June 1923.	15/12/24	A.	Hernial sac present and removed at operation.
35	James A.	10	Lt. I. D. T. & Lt. Ing. H.	May 1923.	15/12/24	A.	Left testicle is slightly larger than right. No hernial sac found at the operation.
36	Charles B.	9	Rt. I. D. T.	Dec. 1923.	15/12/24	A.	Right testicle slightly lower than left. No hernial sac found at the operation.
37	Archie S.	12	Bilateral I. D. T.	Right side, April 1923.	15/12/24	A.	Admitted Jan. 1925 for operation on left side.
38	Leslie P.	15	Lt. I. D. T.	Oct. 1923.	15/12/24	A.	Cremasteric reflex absent. Hernial sac present and excised.
39	Arthur Wm. A.	7	Lt. I. D. T. & Double Ing. H.	March 1923.	15/12/24	A.	Left testicle slightly larger than right. Both hernial sacs removed through a transverse incision.
40	Richard E.	10	Rt. I. D. T.	Nov. 1923.	15/12/24	A.	Cremasteric reflex absent right side, present left.
41	Laurence W.	12	Bilateral I. D. T.	Right side, Feb. 1923. Left side, Aug. 1923.	15/12/24	A. } C. }	Hernial sacs present and excised at operation. Left testicle atrophied. Left testicle could not be felt before operation. It was found in the abdomen and brought down. Post-operative haematoma.
42	S. L. B.	20	Bilateral I. D. T.	Right side, Oct. 1923.	15/12/24	A.	Testicle has increased in size. Hernial sac removed at operation. Operation on left side Nov. 1924.
43	Frank M.	8	Bilateral I. D. T.	Left side, May 1923.	15/12/23	B.	Left testicle is small but of normal consistence and not atrophied. Operation on right side postponed for two or three years to give the transplanted testis a chance of developing, as it probably will.

## A METHOD OF TREATING SEVERE GRADES OF PERITONITIS

By J. GAYMER JONES, M.C., M.S., Surgical Registrar, Guy's Hospital.

ALTHOUGH modern methods have done much to reduce the mortality due to peritonitis, an unduly high death-rate is still associated with the most severe forms of the disease, and it is in the treatment of these forms that the usual methods of drainage are so inadequate. It is not intended to deal, therefore, with those cases in which the surgeon, after gently mopping up the exudate if present, feels that he is justified either in closing the peritoneal cavity, or in expecting a good result if he leaves a tube in for 24 to 48 hours.

In treating the worst cases of peritonitis it is essential to effect free and efficient drainage, and there is no doubt this cannot be done by means of a tube alone, which simply acts as an overflow pipe. A certain amount can be done to help drainage by turning the patient occasionally, but it still remains far short of perfect. This is evidenced by the fact that a rapid flow of exudate frequently follows the removal of the tube, illustrating that even as an overflow pipe it is often inefficient and may prevent drainage.\*

Before proceeding to describe a method which has been used in certain wards of this hospital for some time, it is necessary to state that the word irrigation does not mean the rapid washing out and flooding of the peritoneal cavity at the time of operation. Such a procedure has long been abandoned.

The term as here used means that warm saline solution is allowed to percolate slowly through those areas of the peritoneum to which pus has *already* spread down to the bottom of the pouch of Douglas; from which place it is withdrawn by suction. During this gravitation through the affected areas, the

\* It is desired to emphasise how harmful is the use of large lateral holes in ordinary rubber drainage tubes when used for peritoneal drainage. Most surgeons have seen cases where there has been difficulty in removing the tube owing to the fact that a small portion of the wall of the gut has herniated into one of the holes, and this in spite of the tube having been turned daily. Such a condition is obviously liable to assist in the production of ileus, and the holes are no aid to drainage. Perhaps the best method of applying the ordinary rubber tube is to slit it along its length and to place within it, if necessary, some ribbon gauze as a wick.

saline solution gently washes the pus down to the lowest point and in so doing dilutes any toxins that may be absorbed. That a certain amount of the saline solution is absorbed can be proved by measuring and comparing the amounts introduced and collected. Indeed it is highly likely that the good results depend in part upon this absorption. It might be suggested that absorption of saline solution from a septic area of the peritoneum is harmful, but if one dwells for a moment on the pathological processes involved it must become evident that this is not the case. The circulation of both blood and lymph in the walls of the affected coils of intestine will be improved by the absorption of the fluid, and as this circulation, in bad cases, may be markedly impaired, its improvement, by assisting in the removal of harmful products, will prevent the occurrence of those changes in the gut wall which bring about the condition of paralytic ileus.

The saline solution is introduced by means of Carrol's tubes at the rate of a drop a second (*i.e.* about one pint in  $2\frac{1}{2}$  hours). When the method is used in severe cases of peritonitis following appendicitis, it will commonly be found necessary to use three tubes; they are placed according to the spread of the peritonitis, and this usually indicates the following positions: (1) to the right of the cæcum; (2) in the pelvis; (3) in the left iliac fossa.

The purulent fluid is withdrawn continuously in the following manner. A tube of fairly wide diameter is slit along its length, and within it is placed a narrower tube so as to reach to within half an inch of the lower extremity of the outer tube. So arranged, the tubes are placed to the bottom of the pouch of Douglas or abscess cavity. The upper end of the outer tube just clears the surface of the skin, while the inner tube, which is longer, comes through the dressings and is connected up with a Woulfe's bottle after the patient has been returned to bed. A negative pressure is maintained within the bottle by means of a Sprengel's pump attached to a tap.

The opening in the parietes, through which this series of tubes is placed, is little larger than that necessary for an ordinary drainage tube, and, as will be shown later, hernia is less likely to occur.

The larger tube acts as a well into which the fluid gravitates, and the narrow one withdraws this fluid by means of suction. If the narrow tube projects at all beyond the lower end of the wide one, gut will immediately be sucked on to its end and drainage will cease. An occasional adjustment is therefore necessary, but this can be done without removing the dressings

or bandages. One has only to watch the Woulfe's bottle rapidly filling with pus to realise the immense benefit the patient must be deriving from such a method of treatment.

The time of removal of the tubes is judged by the character of the fluid that is withdrawn into the bottle. When this becomes clear irrigation is no longer necessary. This is most commonly found to be the case in about 24 to 48 hours. When such time has arrived the large tube and the narrower one within it are removed, but one or two disconnected Carrol's tubes are left in for a further 24 hours as drainage tubes; and it is remarkable how well they act in this capacity.

The advantages of the above method may be summarised thus :

(1) The toxic fluid is diluted and rapidly, continuously and effectively withdrawn from the peritoneal cavity instead of remaining to bathe the pelvic coils of intestine and to be in large part absorbed. These patients, therefore, are less likely to develop ileus and lose their toxæmia earlier than would be the case if an ordinary tube were used.

(2) Large quantities of the saline solution are absorbed with resulting benefit to the patients, and the actual absorption improves the circulation in the affected coils of intestine, thereby rendering paralytic ileus even less likely to supervene.

(3) Infection of the abdominal wall is uncommon, as most of the pus is drained away without coming into contact with the fat and muscle; any which reaches these layers is diluted.

(4) As a result of (3), hernia occurs but rarely—and this in those very cases in which it is most prone to do so. The wounds, in fact, heal rapidly, and after a few days there is little to show that there was once a severe peritoneal infection.

For the reasons stated above there can be little doubt that lives can be saved in critical cases and convalescence shortened in others. Residual abscesses have been found to be rare.

This treatment will also be found to be useful in those unusual but distressing cases in which peritonitis develops insidiously after some abdominal operation. These patients are generally desperately ill, and the above treatment may tide them over a very trying period. A small supra-pubic incision can be made in bed under a local anæsthetic with the minimum of disturbance to the patient and the tubes quickly placed in position.

The writer has treated successfully a case of very acute pneumococcal peritonitis by the method described.

There has been a tendency recently to treat peritonitis by indirect methods, such as lymphaticostomy,<sup>1</sup> but these are an admission of failure in the direct treatment of peritonitis, and

they deal only with the toxic absorption that is taking place, completely neglecting the fact that by allowing the peritonitis to be inadequately treated, paralytic ileus is being courted. On the other hand, an ingenious operation has been devised for the treatment of paralytic ileus.<sup>2</sup> This operation, however good it may be when advanced ileus has set in, is again an admission of failure, and it is interesting to note that in the cases quoted in Sampson Handley's paper on the subject, the ileus developed or became worse subsequently to the first operation, and one cannot help feeling that with more efficient treatment of the peritonitis the necessity for the procedure adopted would never have arisen.

If ileus is present or feared pituitary extract ( $\frac{1}{2}$  c.c.) and eserine (gr.  $\frac{1}{160}$ ), as advised by Kerr Cross,<sup>3</sup> should be given. The combination is undoubtedly better than the use of either drug separately, and it should be administered on the first sign of the dread complaint—immediately after operation, if necessary.

In conclusion I wish to thank Mr. F. J. Steward for impressing upon me the advantages of this method which he first described in a lecture printed in the *British Medical Journal* of April 17th, 1920.

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## MENINGITIS OF NASAL ORIGIN

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MENINGITIS of nasal origin occurs as a primary and sole complication of injury to the roof of the nose, as in fracture of the anterior fossa of the skull involving the cribriform plate or as a result of damage to the cribriform plate by direct violence or during intra-nasal operations; more often meningitis is the sequel or end-result of some other intra-cranial complication of nasal disease, such as pachymeningitis, cerebral abscess, cavernous sinus thrombosis, or osteomyelitis of the frontal bone. It may also be the sole complication of diseases of or operations on the nose.

### PATHS OF INFECTION

Gross lesions of the cribriform plate open a direct path from the nasal cavity to the meningeal space, and the course of infection is obvious, especially if there is pus in the nose as a result of accessory sinus disease.

In the absence of such direct path the infection can take one of the following paths :

1. *Infection through bone*, by osteo-myelitis or osteo-phlebitis of the cerebral wall till infection reaches the dura mater (Katz and Blumenfeld). Gerber found that of 87 post-mortem examinations of complications of frontal sinus suppuration 52 showed bone disease and infection by contact; in 6 only was the bone intact. Hinsberg showed microscopically thrombosis of the veins from the mucous membrane of the frontal sinus through the bone to the dura mater. Hajek in a case of ethmoidal suppuration and meningitis found thickening of the subepithelial tissue and streptococci in this and the vessels, though the bone looked fairly normal. This supported Hajek's view of infection by tissue continuity, the bone and dura being infected though they may look normal. Reynolds of Edinburgh has shown microscopically the path of infection to the dura through bone in the case of the sphenoidal sinus. Ortmann in a case of sphenoidal sinus suppuration found diplococci in the mucous membrane of the sinus, in the bone and in the dura. When the disease reaches the dura, pachymeningitis may result and remain dormant for a considerable time, or infection may at once spread through the meningeal space, thus giving rise to fatal diffuse meningitis.

2. *Venous spread*. Microscopically thrombosis may be shown to be responsible for spread of disease through bone, as has just been shown, but infection viâ the venous anastomoses

about the root of the nose is a real danger and no doubt accounts for thrombosis of the cavernous sinus, though in the majority of cases direct spread from the sphenoidal sinus by bone causes this thrombosis, just as is the case with lateral sinus thrombosis from mastoid suppuration. Milligan stated in 1922 that thrombosis of accessory nasal sinus origin took place viâ the ophthalmic veins, and this is recognised as the usual channel. Burger in 1923 agreed, but pointed out that such was not necessarily the case. He stated that the bulk of the venous flow both from the nasal mucosa and the accessory sinuses took place by the spheno-palatine vein to the pterygoid plexus; he quoted cases to support this view. As cavernous sinus thrombosis is only mentioned here as a forerunner of meningitis, the question cannot be pursued further. Lyman mentioned a case seen post mortem of superior longitudinal sinus thrombosis with an abscess on the upper part of the right Rolandic area after an operation on the ethmoid; there was no damage to the roof of ethmoid. Zuckerkandl by injecting the anterior part of the superior longitudinal sinus reached veins in the frontal bone, in the mucous membrane of the frontal sinus and in the back part of nasal mucous membrane, thus showing that infection may pass that way. Diploëtic veins may also convey infection. Direct spread to the meninges from the frontal sinus through the bone is known to occur without any venous thrombosis, and Mullin, after injection of Indian ink into the frontal sinus, found that it had passed straight through the bone into the dura mater. Perhaps this is due to venous spread.

3. *Lymphatic spread* is doubtless a fairly frequent path of infection, especially in the rapid cases after aseptic operations on the nose, e.g. submucous resection or cauterisation of the middle turbinal, and it is stated to be the path taken when meningitis follows the retention of plugs in the nose.

It has been shown that there are lymphatics round the fibres of the olfactory nerve. In addition independent lymphatics pass from the nasal mucous membrane, especially the olfactory portion of it, direct to the meninges. André was able to inject lymph vessels in infants from the sub-arachnoid space to the olfactory mucous membranes, a fact observed also by Schwalbe, Poirier and others.

4. A rare path of infection must be that through *congenital defects* in the posterior wall of the frontal sinus: such cases have been recorded.

#### ORIGIN OF MENINGITIS

The question of meningitis as a sequel to fractures of the interior fossa of the skull will not be considered. In the

presence of accessory sinus suppuration meningitis occurs either spontaneously or as the result of operation, though it may also occur as an accident after "aseptic" operations on the nose. The cases thus divide themselves into two groups: Non-operative and Operative.

*Non-operative cases.* These generally occur in the course of chronic disease, rarely as the result of acute disease. In the great majority of cases there is some other complication present, extradural abscess (pachymeningitis), cerebral abscess, thrombosis of the cavernous or superior longitudinal sinus. The following figures from Katz and Blumenfeld show the relative frequency of the sinuses causing meningitis:

Frontal sinus.	Meningitis (67 cases)	67
	Thrombosis of sup. long. sinus, most with meningitis	7
	Thrombosis of cavernous sinus, most with meningitis	6
Ethmoid	Meningitis (generally a defect in the bony wall)	12
Sphenoid	Meningitis (generally a defect in the bony wall)	31
	Thrombosis of cavernous sinus (often with meningitis)	25
Antrum	Meningitis	2
	Thrombosis of cavernous sinus (Burger's case added)	3

Probably infection of sinuses often escapes notice at post mortem examination in cases of death from "unexplained" meningitis, though no figures are available.

*Operative cases.* This group may conveniently be subdivided into two:—

(a) Those following external operations on the frontal sinus or ethmoid;

(b) those following intra-nasal operations.

(a) The experience with *external operation* for frontal sinus suppuration was at first unfortunate. A number of cases developed meningitis, but it was generally found that this was the result of infection of the bone at operation, osteitis or osteomyelitis or some accident at operation, such as a wound of the dura, damage to the cribriform plate, or to post-operative osteomyelitis of the frontal bone: with improved technique the percentage of cases of meningitis has greatly diminished. Ogston-Luc's operation was discarded for these reasons; Kuhnt's operation gave better results, and Killian's gave good results, though several cases of meningitis are known to have followed it. Howarth's operation gives excellent results, and I have not heard of meningitis after it. It should be noted how much greater is the danger of meningitis after frontal sinus operation than after the radical mastoid operation. On the other hand the dura mater can be wounded without serious results; I have had two cases in which no effects followed such a wound.

(b) The group of cases following *intra-nasal operation* is the one which chiefly interests rhinologists. The number of intra-

nasal operations performed is very large, while the number of cases of meningitis is very small; the percentage of fatalities is probably minute—certainly much less than the percentage of meningitis among a corresponding number of aural cases. Burger in 16 years saw 49 cases of lateral sinus thrombosis, in 3 of which cavernous sinus thrombosis was also present, and 4 cases of cavernous sinus thrombosis of nasal origin. Bruhl noted that among 35,000 ear, nose and throat cases there was only one fatal case of intra-cranial complication from frontal sinus suppuration. In a discussion on the matter Mygind stated that there had been only one fatal case following intra-nasal operations in his Clinic during a number of years. As will be seen later, these figures were very small compared with those from other sources. Jansen in 800 cases of operation saw 3 cases of suppurative meningitis of nasal origin.

At the same time it must be recognised that many fatal accidents are not published. The bulk of cases occurred after operations for the relief of sinus suppuration—that is, the operation was performed in a septic field, but a few followed simple submucous resection of the septum, removal of the interior turbinal and cauterisation of the middle turbinal. In a few cases an operation on the nose lighted up a pre-existing but unsuspected pachymeningitis; thus Watson-Williams performed a left-sided intra-nasal antral operation; the right side was not touched; the patient developed meningitis; at the post-mortem examination a deficiency was found in the roof of the right ethmoid with a patch of pachymeningitis from which the fatal meningitis had started.

Harris records the following case. An officer aged 26, healthy, underwent removal of the anterior end of the right middle turbinal on account of cystic distension. Some years previously he had had a fracture of the nose at base-ball and was incapacitated for two weeks. The fifth day after operation headache and vomiting occurred; the cerebrospinal fluid was cloudy; drowsiness followed and passed into coma and death. Post-mortem examination showed a hole, 5 mm. in diameter, with smooth edges, in the cribriform plate just to the right of the crista galli; the brain and dura mater were adherent to the edges. Obviously an old lesion due to the previous accident, and the meningitis was lighted up by operation.

In some cases the onset of the meningitis is delayed for months. The case published by Gregory is a striking one. In October 1911 a boy of 16 was operated upon for removal of the anterior ends of the middle turbinals and adenoids. At that time he showed no signs of ethmoiditis. In June 1912, nine months later, Gregory saw him in an attack of meningitis,

from which he died in a few days. At the post-mortem examination a hole, 7 mm. by 5 mm., was found in the roof of the left posterior ethmoidal cells, through which a probe passed directly into the left nostril. The cells were full of gelatinous mucus. The meningitis was basal and a pure growth of pneumococcus was obtained.

Almost every variety of intra-nasal operation has been responsible for meningitis.

Katz and Blumenfeld in 1923 stated that intra-nasal operations are the cause of many deaths from meningitis or cavernous sinus thrombosis.

Thus they collected :

After cauterisation of middle turbinal, removal of their anterior ends	
or removal of polypi . . . . .	7 deaths.
After removal of polypi only . . . . .	7 deaths.
After resection of ethmoidal labyrinth . . . . .	12 deaths.

Loeb collected by means of a questionnaire to 700 specialists in America 332 fatalities, of which 125 were due to meningitis, and of these 125, 120 followed nasal operations; this number was made up as follows :

<i>Operation.</i>	<i>No. of deaths.</i>
1. Intra-nasal frontal sinus operation . . . . .	16
2. Probing and irrigation of frontal sinus . . . . .	7
3. Ethmoid operation . . . . .	39
4. Sphenoid operation . . . . .	10
5. Maxillary sinus operation . . . . .	1
6. Removal of polypi . . . . .	13
7. Combined sinus operation . . . . .	1
8. Submucous resection and sinus operation . . . . .	5
9. Resection of middle turbinal . . . . .	15
10. Submucous resection of the septum . . . . .	13
	<hr/> 120

The cause of death was various: damage to the cribriform plate was prominent; some operators saw cerebrospinal fluid flow from the nose at operation. Of nine cases examined post-mortem six showed fracture of the cribriform plate. Leaving plugs in the nose was held to be responsible in some cases.

The following cases were collected from other sources. In Lund's case fatal meningitis occurred a few days after a submucous resection. Pneumococci were found in the nose and the cerebrospinal fluid. Of this case Mygind stated it was the only case of death after a septum operation among the few mishaps in his clinic at Copenhagen.

At a meeting of the Laryngological Section of the Royal Society of Medicine in 1916 a discussion on the accidents after nasal operations was held, the communications being anonymous. The following were quoted, in which meningitis proved fatal:—

A case of submucous resection of the septum on a patient aged 27; death twenty days later. Post mortem pus in the ethmoid and sphenoidal sinuses.

2 cases of ethmoid curetting, death five days later.

1 case of removal of polyp with small piece of bone attached, seen to be cribriform plate. Death soon after.

1 case of intra-nasal operation on the frontal sinus antrum and polypi. Death a few days later. Post mortem, cribriform plate intact, also the dura over it: purulent basal meningitis. In this case infection must have spread by venous or lymphatic paths.

1 case of ethmoid operation followed by meningitis.

1 case of ethmoid operation followed by cavernous sinus thrombosis and meningitis.

3 cases of removal of inferior turbinal followed by meningitis.

To these are to be added the following (the author's):

(a) An officer sustained a through-and-through bullet wound that destroyed both eyes and caused dense adhesions between the septum and ethmoid. Nasal obstruction was so severe that operation to improve matters was undertaken, pieces of what appeared to be ethmoid were removed and bony adhesions divided; meningitis followed soon.

(b) A woman aged 30 suffered from left-sided ethmoiditis; intra-nasal operation was performed and soft putty-like material found in all the ethmoidal cells and sphenoid; she made a good recovery. Two years later she developed a similar condition on the right side; again at intra-nasal operation soft putty-like masses were found in the frontal sinus, ethmoid antrum and sphenoid; all these sinuses were drained. The day following the patient complained of severe headache and vomited; the temperature rose gradually till it was 105° F. on the fourth day; she became comatose and died.

#### PROGNOSIS

Though the prognosis of post-operative meningitis is extremely bad a few cases of recovery have been reported. Gerber quotes a case in which severe headache came on six months after an external operation on the right frontal sinus. The scar was re-opened; the cavity found filled with granulations and a defect in the posterior wall with the dura bulging and congested. The dura was incised, serous fluid came out under great pressure and soaked the dressings for twenty-four hours; ten days later further symptoms of meningitis developed; the dura was more freely opened, with escape of much blood-stained watery fluid, also from the frontal lobe. Recovery.

Piffi had a case of a man with chronic polysinusitis who suddenly developed symptoms of meningitis, vomiting, slow pulse, optic neuritis and stiff neck. Operation through the frontal sinus, dura found under much tension; on incision much clear fluid escaped under considerable pressure and continued to do so for some time. Recovery.

Parry performed a submucous resection; four days later sudden illness with temperature of 105° F.; erysipelas involved the whole face. Maniacal symptoms followed, convulsions, conjugate deviation of head and eyes to the right, squint, rigidity, loss of reflexes and coma. Antistreptococcic serum was administered, 10 c.c. every six hours; improvement in twenty-four hours, convalescent six weeks later.

In Plum's case, after removal of the middle turbinal for lupus, the cribriform plate was probably injured; the nose was plugged; meningitis developed the same evening; the plug was removed; recovery.

I have been fortunate in having had only two fatal cases already quoted, and still more fortunate in having had two recoveries. In the first case the diagnosis is not certain, but the symptoms suggestive: a lady of 36 had right-sided antral suppuration and polypi; an intra-nasal opening was made into the antrum, the polypi removed and exploration of the left antrum revealed clear yellow fluid. Four years later on account of toxic symptoms the posterior ethmoidal cells and sphenoidal sinus were found to contain pus and drained. Sluder's knife was used, and on probing far back it seemed as though the probe went through too far and the blood that escaped seemed watery. For the first four days after operation the patient complained of extremely severe headache, far worse than she had ever had before; the temperature rose each day to at least 100° F. Thereafter the symptoms subsided and recovery followed.

The second case of recovery was after an external frontal sinus operation: in this case meningitis developed some seven weeks after operation on the left frontal sinus and septum; further operation showed extensive extra dural granulations and thrombosis of the vein from the foramen cæcum to the superior longitudinal sinus; repeated lumbar punctures were performed and patient slowly recovered.

#### DIAGNOSIS

The diagnosis of rhinogenic meningitis does not differ materially from that of otogenic origin. The earliest symptom is headache, and, if it was present before on account of the sinus suppuration, it may change its position and become more severe; vomiting, sudden heat flashes, stupor, stiffness of neck,

fever, photophobia; the patient is disquieted, sleepless, restless, the pulse usually fast. Later Kernig's sign is present, the external rectus paralysed, and changes occur in the fundus oculi; then delirium and coma. The changes in the cerebro-spinal fluid are the same as in other forms of infective meningitis. It may, however, be noted that rhinogenic meningitis generally runs an extremely rapid course.

### TREATMENT

Treatment is of little avail when once the disease is established, particularly when an intra-nasal operation was the cause; drainage should be aimed at. The best defence lies in taking all precautions against damage to the cribriform plate during operation and in observing strict asepsis.

Katz gives a few good rules :—

1. Never operate when patient has an acute cold.
2. If possible open all sinuses containing pus at the one sitting.
3. Never plug more than twelve hours.
4. Treat the middle turbinals gently.
5. Avoid the olfactory sulcus.

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# A CLINICAL STUDY OF POLYPI OF THE PROSTATIC URETHRA AND THEIR RELATION TO CHRONIC PROSTATITIS, WITH A REPORT OF THIRTEEN CASES

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THE first recorded case of a benign tumour of the urethra dates from 1607, when Layseau<sup>1</sup> is credited with having removed a papilloma from the urethra of Henri IV of France.

The earliest description of the urethroscopic appearances of these tumours is that of Tarnowsky<sup>2</sup> in 1872, and a few years later Grünfeld<sup>3</sup> collected and described eighteen cases. The great majority of the older reported cases are of papillomata of the anterior urethra, and descriptions of tumours of the prostatic portion of the urethra are much less common.

Bryant<sup>4</sup> in 1893 removed a polyp from the prostatic urethra by perineal incision, a section of which is in the Museum of the Royal College of Surgeons.<sup>21</sup>

During the present century, coincident with the improvement in urethroscopes and urethroscopic technique, many cases of benign tumours of the prostatic urethra have been reported. The majority have been found in association with chronic gonorrhœal urethritis, in which condition urethroscopy reveals lesions varying from slight granulations of the mucous membrane to elongated pedunculated polypi.

A few cases occur in which no signs of gonorrhœa were discovered nor a history admitted, examples being quoted by Thomas,<sup>5</sup> Player and Mathé,<sup>6</sup> and Randall.<sup>7</sup> Polypi in this situation are also found in association with senile enlargement of the prostate gland and in the prostatic capsule after prostatectomy, as reported by Randall,<sup>7</sup> McGowan,<sup>8</sup> and Culver.<sup>20</sup>

Polypi of a somewhat similar type have been found by Kreutzmann<sup>9</sup> in the female urethra associated with chronic cystitis; in many of his cases there was no evidence or history of gonorrhœa.

Occasionally polypi have been found in animals—a good example being a large pedunculated polyp in the prostatic urethra of an ox, which is preserved in the Museum of the Royal College of Surgeons.<sup>10</sup>

The general pathological classification of these urethral tumours has been the subject of discussion for many years. Lewis and Mark <sup>11</sup> classify them into

- (1) Vascular polypi.
- (2) Fibromata.
- (3) Fibro-myomata.
- (4) Fibro-myxomata.

Player and Mathé <sup>6</sup> discuss the microscopical appearances in eight cases, dividing them into two groups, fibrous and glandular, and laying stress upon the evidence of inflammatory changes in all cases. Randall <sup>7</sup> classifies his cases into fibrous, villous, and glandular polypi, and considers that they are very similar to the so-called mucous polyps occurring elsewhere in the body. Thomson-Walker <sup>12</sup> states they may be separated into four groups :

- (1) Fibroma.
- (2) Adenoma.
- (3) Myoma.
- (4) Fibro-myoma.

McKenna <sup>13</sup> differentiates between those of an inflammatory nature and benign growths :

- (1) Granulations and Polypi.
- (2) Benign Growths. . . .  $\left\{ \begin{array}{l} \text{Myoma.} \\ \text{Fibroma.} \end{array} \right.$

Although these various classifications are not in complete agreement, it is generally considered that a large proportion of polypi in the prostatic urethra are of an inflammatory nature, although genuine benign new growths have been found.

During the routine examination of the prostatic urethra in chronic urethritis, the pathological lesions that may be discovered vary considerably in appearance, from the usual slight effects of a persistent inflammation of low degree to the grosser conditions of infiltrated areas in the mucous membrane, granulations, polypoid projections, cysts, and pedunculated polypi.

The general characteristics of these various lesions are in keeping with the effects of a chronic inflammatory process of long duration, and as in other mucous tracts the lesion may be general or solitary.

This article is confined to a consideration of those conditions which presented a definite pedunculated tumour of a type clinically comparable to the tumours found elsewhere in the body, which are commonly known by the ill-defined term of polyp.

All the cases in this series were discovered on urethroscopic examination of the prostatic urethra in patients suffering from chronic urethritis, usually of a gonorrhœal origin.

The general type of polyp discovered appeared as an elongated pedunculated swelling of small size, usually single, arising from the mucous membrane in the vicinity of the verumontanum and characterised by its mobility and vascularity.

### SYMPTOMS

1. *Hæmaturia*.—In contradistinction to many previously reported cases, hæmaturia did not occur in any case of this series.

Isolated cases with profuse hæmaturia have been reported by Lewin,<sup>14</sup> Thomas,<sup>5</sup> McGowan,<sup>8</sup> Davis,<sup>15</sup> and others. These cases were reported on account of the marked hæmorrhage and are not part of a series. If the symptoms of a series of cases are examined it will be found that hæmaturia is infrequent.

Player and Mathé<sup>6</sup> in an analysis of sixty-eight cases report hæmaturia once only. Randall<sup>7</sup> also in his series of twelve cases reports hæmorrhage on only one occasion, and comments upon its rarity. Hæmorrhage from polypi in the posterior urethra is therefore distinctly uncommon as a symptom *per se*, although it may follow the passing of instruments or application of strong chemical solutions.

2. *Pain associated with Polypi*.—In this series of cases pain was rarely complained of and when present was of the nature of perineal irritation or discomfort.

This comparative absence of pain appears to be unusual, but may be partly accounted for by the fact that in many other reported cases urethroscopic examination was undertaken on account of the painful sensation, whereas in this series the examination was undertaken as a routine.

Player and Mathé<sup>6</sup> in a collection of sixty-eight cases of polypi report pain to be a frequent symptom and state the following proportions and analysis :

Perineal discomfort . . . . .	57
Lumbo-sacral backache . . . . .	54
Inguinal pain . . . . .	20
Hypogastric pain . . . . .	13
Testicular pain . . . . .	9
Painful ejaculations . . . . .	8
Tenesmus . . . . .	10

Randall<sup>7</sup> reports pain of varying character in six out of fourteen cases.

Pain may be set up by polypi of large size causing interference with micturition, as in a case reported by Davis<sup>15</sup> which culminated in retention. Dragging of the pedicle may be a cause of pain during micturition; a marked stinging pain associated with a long polypus has been reported by Randall.<sup>7</sup>

Small polypi, however, appear to cause no pain unless accompanied by inflammatory lesions of the prostatic urethra, and such conditions are the rule rather than the exception. The majority of symptoms appear to be due to the associated inflammatory conditions of the urethra, prostate gland or seminal vesicles. Randall<sup>7</sup> reports two such cases, in which the prominent symptoms were in one supra-pubic pain and backache in the other; it is possible that both of these symptoms were due to the associated prostatitis.

Player and Mathé,<sup>6</sup> whose analysis of types of pain appears above, consider that chronic prostatitis and vesiculitis are frequently present.

If, however, as in this series, the prostatic urethra is examined as a routine in cases of chronic urethritis, it will be found that polypi will be discovered in cases which are entirely free from painful sensations.

3. *Disturbances of a Sexual Nature.*—Although disturbances of a sexual nature were not complained of by any case in this series, they are not infrequently met with in association with polypi of the prostatic urethra; according to McKenna<sup>13</sup> and Randall<sup>7</sup> they are a frequent symptom.

Impotence, loss of sexual desire, and increasing frequency of nocturnal emissions are the most frequent disorders, whilst premature ejaculations and hæmorrhagic seminal emissions have also been noted. Impotence especially appears to be frequent and was noted by Player and Mathé<sup>6</sup> in eight cases, and has also been reported by McGowan,<sup>8</sup> Randall,<sup>7</sup> and Fürbringer.<sup>16</sup>

Here again it is difficult to dissociate symptoms produced by the presence of polypi from those due to concurrent inflammatory lesions of the prostate gland and seminal vesicles.

Player and Mathé<sup>6</sup> report three cases of polypi associated with chronic prostatitis and vesiculitis, in which the sexual symptoms present were diminishing sexual power in two cases and frequent nocturnal emissions in one; the latter symptom was probably due to the vesiculitis.

The symptoms in general are more likely to be due to the associated inflammatory conditions than to the polypi themselves.

The following reports of thirteen cases illustrate the close association of such polypi with inflammatory lesions of the

prostate gland. It is regretted that the case records of some of the earlier cases are rather meagre; at the time the cases were under observation the association of the two conditions was not realised. In other cases the attendances were so irregular that the clinical history is unavoidably incomplete.

*Case 1. Æt. 43.*—A sailor who on first attendance gave a history of having suffered from persistent morning gleet for one and a half years following an attack of gonorrhœa. During this period he had had three relapses of profuse urethral discharge, and had been undergoing treatment at a naval hospital by irrigation and prostatic massage.

On first examination a gleet was present; his urine contained a few purulent flakes in an otherwise clear fluid: the flakes on examination were found to contain pus cells but no gonococci.

Examination of the prostatic secretion after massage revealed pus cells, but no gonococci were found. Treatment by digital prostatic massage was carried out regularly for two months; at the end of this period cystoscopy showed no abnormality, but urethroscopic examination revealed a large fleshy cauliflower polyp the base of which was attached to the verumontanum.

The polyp was destroyed by the application of diathermy through a posterior urethroscope, under visual control, and two months later no sign of polyp could be found. The prostatitis, however, persisted for a further three months, at the end of which period the case ceased attending.

It will be seen that the prostatitis dated back to about twenty months prior to the discovery of the polyp, and was still found to be present five months after total destruction of the polyp. No gonococci were found in this case at any time during his attendance over a period of six months.

*Case 2.*—This patient had suffered from a slight urethral discharge for seventeen months following an attack of acute gonorrhœa. No other symptoms were complained of, and the urine contained a few fine threads only.

The prostate gland and seminal vesicles were normal on palpation, and the prostatic secretion was also microscopically normal.

No abnormality other than slight injection of the mucous membrane was revealed by cystoscopy or anterior urethroscopy. On urethroscopic examination of the prostatic urethra a polypoid mass was found attached to the lower extremity of the verumontanum.

The patient attended on a few further occasions and was not seen again.

There appears to be no evidence of chronic prostatitis in this case.

*Case 3. Æt. 31.*—An acute attack of gonorrhœa began in July 1919 whilst he was in the army; treatment was carried

out at a military hospital over a period of six months, at the end of which time he was discharged with a non-infectious gleet.

He first came under observation in December 1919, complaining of gleet; his urine contained purulent threads, but no gonococci were found. Treatment by prostatic massage and irrigation was instituted. In May 1920 he complained for a few days only of slight pain at the end of micturition. In July 1920 his gleet had subsided and his prostatic secretion was found to be normal on three occasions.

In September 1920 posterior urethroscopy revealed two small pedunculated polyps, with slender stalks, attached, one on either side, to the lateral walls of the verumontanum; they were treated by the application of silver nitrate 2 per cent. During the next two months his prostatic secretion was found to be normal on four separate occasions.

In January 1921 a small polypus was found on the summit of the verumontanum, which was again treated with silver nitrate.

Urethroscopy performed six weeks later showed a long polypus attached to the right side of the verumontanum, which was treated with silver nitrate. Six months subsequently, anterior and posterior urethroscopies showed a healthy urethra and no trace of polypi. Cystoscopy showed slight enlargement of the prostate gland and dilated venules on its vesical surface. In January 1922, a few months later, his gleet had cleared, the urine was perfectly clear, but the prostatic secretion was found to contain pus cells, although this was not confirmed on two subsequent similar examinations.

The evidence of any chronic prostatitis in this case is doubtful.

*Case 4.*—First attended in June 1920 for a urethral discharge of three months' duration. In August 1920 he still suffered from chronic urethritis; urethroscopy showed a slight infiltration in the bulb and the prostatic secretion contained pus cells, on numerous occasions, from this time up to April 1921. During this period there were no symptoms beyond a gleet. Treatment was carried out by prostatic massage and irrigations. Posterior urethroscopy in April 1921 showed a small grape-like cluster of minute polypi on the verumontanum, which was treated by the application of silver nitrate 10 per cent. on two occasions.

No further prostatic massage was carried out, and the prostatic secretion was found to be clear of pus cells and sterile on culture in October 1921.

In this case the chronic prostatitis appeared to clear up after dealing with the polypi.

*Case 5. Æt. 23.*—Suffered from acute gonorrhœa in February 1920, followed in March by symptoms of an acute posterior urethritis and prostatitis. In July 1920 he was suffering from a slight gleet only which gave no symptoms. Examination

of the prostatic urethra revealed three minute polypi attached to the summit of the verumontanum in the mid-line. These were treated by instillations into the prostatic urethra of silver nitrate 3 per cent. The gleet had ceased by December 1920, but the prostatic secretion was purulent on two examinations during the month.

In January 1921 the prostatic secretion became normal and remained normal on two subsequent examinations. At this stage urethroscopy showed one polyp only, situated on the left side of the verumontanum.

In April 1921 his prostatic secretion was normal, his urine perfectly clear, and the polypus, which had been treated with instillations of silver nitrate 3 per cent., was now very small. He was seen again in November 1921, when his urine was perfectly clear and there were no symptoms. Examination of the prostatic urethra showed no trace of polypi; the verumontanum was hypertrophied. He reported again in 1923, being free from all symptoms and his urine quite clear.

*Case 6.*—Attended in March 1924 with acute gonorrhœal urethritis of three days' duration. There was no previous history of gonorrhœa. In June 1924 he had an acute epididymitis which did not subside in the usual way owing to a concomitant acute hydrocele, which was relieved by needling: the hydrocele fluid was not purulent and contained no gonococci. Some swelling of the epididymis was still manifest in August 1924, but had resolved by October 1924. The prostatic secretion was found to be purulent in November 1924, and later in this month urethroscopy of the prostatic urethra showed a large fleshy polypus which filled the lumen of the urethroscopic tube. It was stalked and moved freely in the irrigating fluid, but its attachment could not be seen. The verumontanum could not be seen, but was hidden, probably immediately above the polyp. A slight gleet was the only symptom complained of at this period.

Treatment was carried out by prostatic massage and instillations to the prostatic urethra of silver nitrate 5 per cent.

In January 1925 there was a recurrence of discharge containing many gonococci; this subsided to a slight gleet by February 1925; the prostatic secretion, however, was still purulent.

Posterior urethroscopy in February 1925 showed a large swollen verumontanum but no trace of a polypus. Gonococci were still present in the urethral discharge a few days later, and the prostatic secretion was still purulent in March 1925.

*Case 7.* *Æt.* 26.—Attended for a first attack of gonorrhœa, with a discharge of five days' duration. Under treatment the discharge had ceased and urine became clear at end of a month, but a few days later there was a recurrence of discharge which lasted for a week.

A week later, six weeks from the day of first attendance,

the prostatic secretion was found to be purulent and treatment by massage was instituted.

Posterior urethroscopy, at the end of six months from the onset of the disease, showed a tiny pedunculated polyp attached to the centre of the verumontanum.

The prostatic secretion remained slightly purulent for a further period of three months and then cleared up.

*Case 8. Æt. 28.*—Previous attack of gonorrhœa three years ago. Attended for an attack of acute gonorrhœa, which was complicated a few weeks later by an acute prostatitis, but no involvement of the seminal vesicles. This subsided under treatment, leaving a persistent gleet. Three months later his prostatic secretion was still purulent. Two months still later posterior urethroscopy revealed two tiny sessile polypi situated on the summit of the verumontanum.

*Case 9. Æt. 28.*—First attack of gonorrhœa in August 1920, which became chronic and resulted in a slight infiltration in the anterior urethra. This was treated by dilatation, and in April 1921 posterior urethroscopy revealed a large pedunculated polyp attached to the upper part of the crest of the verumontanum.

The polyp was still present in November 1921. There is no record of any examination of the prostatic fluid in this case.

*Case 10. Æt. 27.*—First attended September 1924, with a sub-acute urethritis. The discharge cleared up in a few days under treatment and no gonococci were found. There was no previous history of gonorrhœa. In November 1924 there was no discharge and urine was clear, but prostatic secretion was found to be purulent, and the prostate gland was enlarged and nodular and hard. Posterior urethroscopy showed a large mobile fleshy polyp presenting in the lower part of the prostatic urethra below the verumontanum. This was removed by grasping with forceps and twisting off. Subsequent section and examination proved it to be of an inflammatory nature. The prostatic secretion was found to be still purulent one week later.

*Case 11. Æt. 54.*—First attack of gonorrhœa sixteen years ago; for four years prior to coming under observation had suffered from frequency, backache, and irritation in the perineum. There was no urethral discharge, and urine was quite clear; digital examination of the prostate gland and seminal vesicles was normal; cystoscopy showed a slight bilateral enlargement of the prostate gland.

Anterior urethroscopy revealed no abnormalities; posterior urethroscopy showed a small cockcomb projection of a warty type attached by a thick stalk to the upper left quadrant of the surface of an enlarged congested verumontanum. Treatment was carried out by applications of silver nitrate solution and prostatic massage. The prostatic secretion was purulent



at the time of discovery of the polyp and was still purulent three months later.

*Case 12. Æt. 32.*—Suffered from gonorrhœa twelve years ago. In 1920 reported for a slight urethritis in which no gonococci were found; the discharge cleared up in a week, and urine became clear.

Urethroscopy showed a normal anterior urethra but an inflamed prostatic urethra, and prostatic massage yielded a purulent secretion.

In April 1921 occasional gleet persisted, and urethroscopy revealed an enlarged injected verumontanum, which was treated with a few applications of silver nitrate (1—5 per cent.).

Occasional gleet persisted in the first half of 1922, when again the prostatic secretion was found to be purulent.

This case did not come under observation again until October 1924, a period of two years; the prostatic secretion was still purulent and gleet was complained of.

Urethroscopy showed a normal anterior urethra, but in the prostatic urethra a polypus of large size was found immediately above the verumontanum.

The polyp projected downwards along the axis of the urethra and was composed of gelatinous material with scattered fleshy nodules. The stalk and area of attachment were not seen.

The prostatic secretion was still purulent a month later. After regular prostatic massage and local treatment with silver nitrate (5 per cent.) the polyp was found to have disappeared in March 1925; the verumontanum was still enlarged and inflamed.

*Case 13. Æt. 20.*—Attended for first attack of gonorrhœa in September 1920, and after routine irrigation treatment was apparently well in December, but the prostatic secretion was found to contain both pus cells and gonococci.

Treatment by prostatic massage was continued up to March 1921, when posterior urethroscopy revealed a polypus of cauliflower type attached by a thick stalk to the centre of the verumontanum. This was treated by applications of silver nitrate and no trace of polyp could be seen in August 1921, at which time urine was clear and symptoms entirely absent.

He did not attend again for about a year, at which time there was no discharge and urine was clear. In October 1922 he attended again with a recurrence of discharge in which gonococci were found. Posterior urethroscopy showed a polyp of similar type attached by a thick short stalk in a similar situation. The prostatic secretion was found to be purulent but no gonococci were reported.

He attended at irregular intervals during 1923 and was treated with prostatic massage and irrigation, but no topical treatment for the polypus was carried out.

He reappeared in June 1924 with a recurrence of discharge, gonococci still being present, which cleared up quickly under

irrigation for a while, to reappear once more in November 1924.

Prostatic secretion was still purulent in December 1924.

Posterior urethroscopy, February 1925, showed no trace of polypi. The verumontanum was large and of mulberry type but showed no sign of active inflammation.

It will be noted that in this case the second polyp disappeared under treatment applied for the prostatic infection alone, and without any local treatment to the polyp itself.

#### THE ASSOCIATION OF POLYPI WITH CHRONIC INFECTION OF THE PROSTATE GLAND

The association of polypoid growths in the prostatic urethra with chronic posterior urethritis has long been noted, and their occasional association with chronic infection of the accessory sexual glands has previously been recorded by some few observers, although in the majority of reported cases of polypi the condition of the prostate gland and seminal vesicles is not referred to. Randall<sup>7</sup> in a series of twelve cases of polypi found chronic prostatitis present in three cases, but does not discuss the nature of this association. Player and Mathé,<sup>6</sup> who collected sixty-eight cases, reported seven in which chronic prostatitis and chronic vesiculitis were present, but the frequency of this complication in the remainder of the cases is not given, although the authors consider the association to be frequent.

McKenna<sup>13</sup> reported one case of a polypus associated with a prostatic abscess which had previously ruptured into the urethra.

That urethral polypi are not invariably associated with chronic prostatitis is obvious from their occurrence in the female urethra. Kreutzmann,<sup>9</sup> in a collection of forty cases in the female, stated that they were commonly associated with chronic infection in the urethra or bladder: 50 per cent. of his cases suffered from chronic cystitis.

The usual position of these tumours differs from those in the male in that they were situated immediately outside the vesical sphincter, or on the anterior or lateral walls, and not on the floor of the urethra.

Their general appearance also differs from that of the polypi in the male, inasmuch as they are small sessile tumours of a warty type rather than pedunculated polypi.

In the series of cases reported here the incidence of chronic prostatitis is strikingly high; out of thirteen cases chronic prostatitis was found to be present in ten, or 77 per cent.

In one case there is no record of any examination of the

prostate gland or its secretion, so that if this case is omitted in arriving at the incidence, the percentage in the remaining twelve will be found to be 83 per cent.

It is evident that the association of the two conditions is a common one, and it is probable that they are interdependent.

#### *The Duration of Chronic Prostatitis prior to the Discovery of Polypi*

Chronic prostatitis had existed in the cases of this series for periods varying from two years to one month prior to the discovery of the polyp. In several cases, from the history of recurrences, it is probable that chronic prostatitis had existed as long as four or five years.

This long history has been noted in other cases of polypi: Randall<sup>7</sup> refers to one case in which the chronic prostatitis was of five years' duration, and Player and Mathé<sup>6</sup> quote two cases in which the condition had been present for eighteen years.

Although it is not possible to state the length of time the polyp has been present in all these cases, it is most probable that the chronic infection of the prostate gland had usually existed for a long time prior to the formation of the polyp. Chronic prostatitis is a common complication of gonorrhœa, whereas polypi are rare, and chronic prostatitis is a condition characterised by its long duration and resistance to treatment. Again, prostatitis is an early complication of gonorrhœa, whereas polypi are rarely found until late.

#### *The Recurrence of Polypi*

The recurrence of polypi in the prostatic urethra has been noted by Player and Mathé,<sup>6</sup> who reported two cases, but the presence or absence of chronic prostatitis was not stated.

Recurrences were met with in three cases of this series; in two cases associated with chronic prostatitis, in one the evidence of chronic prostatitis was inconclusive.

*Case 3.*—As will be seen from the history given, two polypi were discovered in September 1920, situated one on either side of the verumontanum, which were treated by the application of silver nitrate. Five months later these polypi were found to have disappeared, but a very small polyp was seen on the summit of the verumontanum, to which silver nitrate was applied. Six weeks later a large polyp was seen attached to the right side of the verumontanum. As already noted, the evidence of chronic prostatitis in this case was not conclusive.

*Case 5.*—Onset of gonorrhœa was followed a few weeks later by acute posterior urethritis and prostatitis which became

chronic. Five months later, three slender polypi were found attached to the summit of the verumontanum, which were treated with local applications of silver nitrate. Chronic prostatitis was still present six months later, at which time the original polypi were found to have disappeared, but another was found attached to the left side of the verumontanum.

*Case 13.*—In March 1921, after a chronic prostatitis of nine months' duration, a polyp of cauliflower type was found attached to the centre of the verumontanum. No polypi could be seen in August 1921, but in October 1922 a polyp of similar type and position was discovered. Chronic prostatitis was still present and persisted up to December 1924. No further polypi were found up to January 1925.

#### *Location of Attachment of Polypi*

In this series and in the great majority of previously reported cases the common position for the attachment of polypi is to the floor of the prostatic urethra, either to some part of the verumontanum or in the lateral sulcus—the prostatic sinus—on either side. It will be remembered that the openings of the common ejaculatory ducts are situated on the summit of the verumontanum in the vicinity of the prostatic utricle; the openings of the numerous prostatic ducts are situated in the prostatic sinuses.

The common position of polypi is found to be in the region of the openings of the ducts of the accessory sexual glands. The frequency of the association of polypi in this situation with chronic infection of the prostatic urethra and of the accessory glands has already been indicated both in this and in other reported collections, and it is reasonable to suspect there is some causal relationship between the two conditions. Several cases have been reported which illustrate the connection in a striking manner. Randall<sup>7</sup> reports a case of a polyp which was attached inside the mouth of the prostatic utricle—a frequent position for the openings of the ejaculatory ducts—associated with chronic vesiculitis and epididymitis.

In another case reported by McKenna<sup>13</sup> a papilloma was attached to the right side of the verumontanum overlying the orifice formed by the rupture of a prostatic abscess. Luys<sup>17</sup> removed a polyp from the prostatic urethra which he considered owed its origin to chronic prostatitis.

Polypi are sometimes found on the lateral walls or roof of the prostatic urethra, although these situations are uncommon. Thomas<sup>5</sup> and Randall<sup>7</sup> reported polypi in these positions.

*Analogy to Nasal and Aural Polypi*

The inflammatory nature and general appearance of these polypi present a close analogy to the well-known polypi frequently found in the nasal fossæ or external auditory meatus and middle ear.

Polypi in these situations are of fibromatous or fibromyxomatous type. They are generally and widely considered to be of an inflammatory nature and to be constantly associated with, and due to, prolonged pre-existing inflammatory conditions in the accessory nasal sinuses or in the middle ear. The removal of such polypi is frequently followed by a recurrence as long as the underlying infection persists.

*The Persistence of Gonococci in Association with Polypi*

At the time of discovery of the polypi and during their known presence the associated gleet was not of a gonococcal nature in the majority of cases in this series. In eight out of the thirteen cases no gonococci were found at any time during their attendance, although all except one gave a definite history of gonorrhœa prior to coming under observation. In three of the cases no gonococci could be found after the first few weeks of treatment, and not less than six months prior to the discovery of the polypi. In two cases polypi and a discharge containing gonococci were present at the same time; in both chronic prostatitis was present.

*Treatment*

The general observations made on these cases make it clear that treatment should not be directed to the polypi alone. The presence or absence of infection in the accessory sexual glands and their ducts must be ascertained and appropriate treatment instituted.

The majority of polypi are of small size and are amenable to treatment by the application of caustics such as silver nitrate. This may be applied through a posterior urethroscope or instilled into the posterior urethra by means of a syringe of the Ultzmann type. For larger polypi the cautery or diathermy through a posterior urethroscope is more thorough. Some polypi may be grasped with a urethral forceps and removed by twisting off, as in Case 10, and sections subsequently made. The bleeding is very slight.

## THE CAUSAL RELATIONSHIP BETWEEN THE POLYPI IN THE PROSTATIC URETHRA AND CHRONIC PROSTATITIS

The possibility of infection of the prostate gland from polypi has recently been indicated by Player and Mathé,<sup>19</sup> who found in several of their cases that the symptoms of chronic prostatitis did not clear up until the destruction of the polypi. They also consider that these growths can act as a source of irritation and cause re-infection of the prostate gland and seminal vesicles.

Luys<sup>17</sup> quotes one case in which the chronic prostatitis and vesiculitis cleared up after destruction of a polypus.

Morrow<sup>18</sup> reports three cases of a similar nature, and Case 4 of this series also provides a similar sequence in which the chronic prostatitis was found to have disappeared after treatment of the polypus.

This view of the relationship appears to be generally held, but several cases in this series point to the occurrence and recurrence of polypi as being due to a persistent underlying chronic prostatitis. Such chronic prostatitis can be found to exist prior to, and subsequent to, the presence of polypi as illustrated by Cases 1, 3, 6, 7, and 13.

Observations have been made illustrating the close connection between these two conditions, both in this series and in series reported by others. From the clinical, the anatomical, and the pathological aspects the accumulated evidence points to the underlying chronic prostatitis as being a probable causal factor in the production of polypi.

On the clinical side there are several notable points.

1. The common but not invariable association of the two conditions in this series and in other collected cases.

2. The long duration of the chronic infection of the prostate gland in many of the cases, prior to the finding of polypi and its persistence after destruction of the polypi.

3. The recurrence of polypi in cases of persistent chronic infection of the prostate gland.

4. The disappearance of polypi under treatment of the chronic prostatitis alone as in Case 13.

From the pathological side there is abundant evidence that inflammatory changes in these polypi are common and that they are frequently of an inflammatory origin.

The anatomical position of these polypi is closely related to the orifices of the prostatic and ejaculatory ducts.

Furthermore, polypi of analogous type in the nose and ear are almost invariably inflammatory in origin and due to persistent underlying inflammatory processes.

It may therefore be considered that the majority of polypi in this region are of an inflammatory nature and are frequently the result of the continual presence of a purulent prostatic secretion from a chronic infection of the prostate gland. It is probable that chronic infection of the seminal vesicles may take a similar part in the formation of some polypi, although there is no evidence of such a sequence in the cases of the series.

#### CONCLUSIONS

Polypi in the prostatic urethra are commonly associated with a chronic prostatitis, which may exist prior to the formation of polypi and persist after their destruction.

Such polypi are most frequently of an inflammatory nature and analogous to polypi in other regions of the body.

The cause of these polypi is frequently due to the underlying chronic prostatitis.

Treatment directed to the polypi only is inefficient unless the chronic prostatitis is also subjected to treatment.

I am indebted to Professor Adrian Stokes for the histological report in Case 10.

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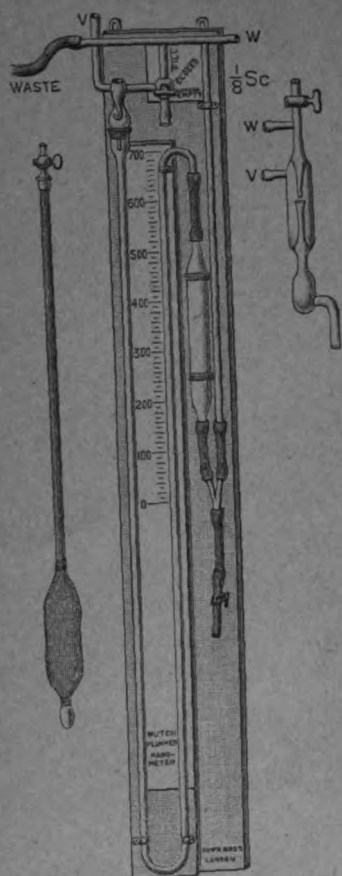
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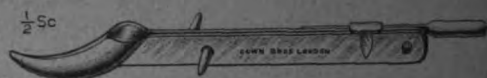
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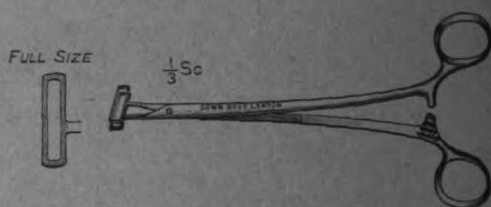


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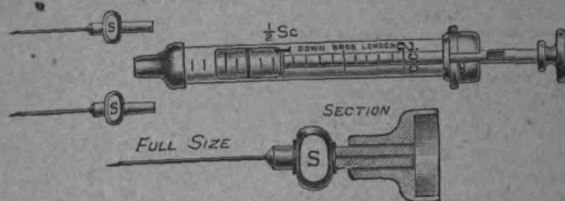
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Keats' Cottage.



Mr. Hammond's house in 1925.

## KEATS AS A MEDICAL STUDENT

By SIR WILLIAM HALE-WHITE, K.B.E., M.D., Consulting Physician to Guy's Hospital.

So much has been written about Keats and such an exhaustive book on him has recently appeared \* that I should not have dared to write of him, were it not that I have carefully read his notes of lectures.

John Keats was born in the last days of October 1795. In 1803 he went to John Clarke's School at Enfield; he left there midsummer 1811, when not quite sixteen, and was then apprenticed to Mr. Thomas Hammond, surgeon, Church Street, Edmonton, being bound for five years.† This was convenient, for he had previously been brought up in his grandmother's house,‡ which was in the same street, and she did not die till 1814. As Edmonton is only two miles from Enfield, Keats often walked to see and to talk of poetry with his friend Clarke at his old school. Hammond must have had a fair practice, for he kept two apprentices, but we have no record of the medical work of either while with him. He let Keats off the last year of the term for which he was bound. Consequently Keats left him at the end of the summer of 1815.

In the Medical School Office at Guy's Hospital are three books connected with Keats. One is that in which new students wrote their names. Some thief has cut out that of Keats for the sake of the autograph. Another is entitled "Guy's Hospital. Entry of Physicians' and Surgeons' Pupils and Dressers, 1814-1827." On p. 29 we read, "Oct. 1st, 1815. John Keats (No. 57), 6 months. Place of Education, Mr. T. Hammond, Edmonton. Office fee £1 2s." In the same book on a later page headed "Dressers to the surgeons entered at Guy's Hospital" is this entry: "1816, March 3, John Keats under

\* *John Keats*, by Amy Lowell, 1925.

† Hammond's house is still occupied by a doctor. It is No. 7 Church Street and is only a few yards from Lamb's cottage on the same side of the road. No. 7 is now known as St. Winifred's; formerly it was called Ten Tree Hall, and in Keats' time, Wilston. Judging by its present appearance, it looks now as it did then, except that an east wing has been added. In the grounds a little to the east of this wing is a tiny dilapidated cottage, known locally as Keats' Cottage, in which Keats was housed. For permission to take the photographs reproduced in the Frontispiece I have to thank Dr. M. P. Menon.

‡ Now pulled down.

Mr. Lucas. Time 12 mths, from Edmonton, £1 1s." The third book is entitled "Surgeons Pupils of Guy's and St. Thomas' Hospitals from Jan. 1812 to separation March 1825"; under the heading "Guy's Hospl. Oct. Division" are two entries: "Oct. 1815, John Keats, £25 4s.," "Oct. 29, 1815, Mr. Jn. Keats returned to him £6 6s., he becoming a dresser." Oct. 1st, 1815, was a Sunday; Keats therefore first came to Guy's on Oct. 2nd, 1815.

The Hampstead Public Library contains the Dilke collection of Keats' books and manuscripts. Among these is a note-book, in which he made notes on anatomy and physiology. As far as I know this has never been fully examined, so, through Mr. Doubleday, the Librarian, I applied to the Committee of the Hampstead Library to have the book photographed and was kindly granted permission, for which I gladly thank the Committee. I have carefully read the photographic copy, one example of which is now in the Library of the Royal Society of Medicine.

The note-book itself is a green-grey colour, small in size and much worn. On the first page is the name John Keats, in his own handwriting, a few scrawls and the figures 425. There is nothing to show to what they refer. The last page has in the top left corner 2/2. If this was the price of the book, it was very dear. There are two little drawings and the Dilke book-plate. The arrangement of the notes is confusing. The twenty-four left-hand pages following that on which is his name are occupied with notes on descriptive anatomy, written so large, so neatly and so well spaced for arresting the attention that they cannot have been taken down at a lecture, but appear to have been made at the writer's leisure.

Turning to the notes on physiology, the second right-hand page is headed "Lectr. 1st," the third right page, "Lectr. 2nd," the fourth "Lectr. 3," but "Lectr. 4" is on the right-hand page opposite the name John Keats. We know that these are notes of a series of physiological lectures, for the first line on this page is "Lectr. 4. Arteries continued from page 3. Physiology." This lecture continues at the other end of the book, on the first right-hand page, which is headed "Lectr. 4 continued." The next left-hand page is headed "Lectr. 5"; the following five right and left pages are taken up with this series of lectures up to and including the eighth. There are no notes of the ninth lecture, but those of the next three are full and these notes end with the twelfth. Then there follow, on three pages in small handwriting, some notes on the composition of bone, on ligaments and on cartilage. Between the

manuscript at either end of the book there are a hundred and eighteen blank pages. The writing of the notes of the lectures on physiology is very small, very close, not spaced and sometimes continued vertically and horizontally on the margins and on the opposite page containing the anatomical notes. The contrast between the large writing of the neatly spaced anatomical notes and the small writing of the closely written physiological notes is striking and has led Sidney Colvin \* to speak of "a note-book in which some other student has begun to put down anatomy notes and Keats has followed." But when I compared the two sets of notes I came to the conclusion that they were in the same handwriting—that of Keats. For example, the formation of the capitals is the same, the *ys* are similar and the way the letters are run together is alike. To confirm this opinion I called on Mr. Doubleday; we compared these notes with the many letters in the Dilke collection. Mr. Doubleday had no doubt that the two sets of notes were both in Keats' handwriting. Finally I took the notes to Mr. T. J. Wise; he kindly examined them, compared them with specimens of Keats' handwriting of the same date in his collection and said that, without doubt, all the notes were written by Keats. Keats' own letters have a diversity of general appearance quite as great as that between the two sets of notes, depending on whether he writes slowly, the spacing, and the pen he uses. In his letters, as in these notes, he was accustomed to write over the margins, indeed he wrote poems on the fly-leaves of books he was reading.

The anatomical notes, which deal with osteology, were made before the physiological, for these, on several pages, are crammed close round and even between the lines of the first. The anatomy is extremely elementary, and the facts are tabulated as much as possible, *e.g.* we have a list of the bones at the base of the skull. Sometimes, on the pages opposite to the osteology, we find surgical notes. Thus a whole page is occupied with close writing describing the treatment for fracture of the ilium: "Mr. C.," whom we shall presently see was Astley Cooper, "has never seen a fracture of the ischium alone," "he has seen a fracture of the pubis do well, if the bladder is not injured." We are told how to tell an inguinal from a femoral hernia, of the dislocations of the hip, of the differences between the male and female pelvis and in operating on the thorax to avoid the intercostal artery. Opposite the page enumerating the bones of the hand and foot are many surgical notes which are continued in between the list of the bones. Fracture of the neck

\* *John Keats*, p. 33, 1917.



of the femur occurs in elderly people: "Mr. C. says that in this case no union ever takes place. Mr. Abernethy is, however, of a contrary opinion. Mr. C. thinks that a fracture through the trochanter major has been mistaken for a fracture through the neck." Club foot is carefully described. "The cause Mr. C. thinks is an unnatural shortening of the gastrocnemius and tendo Achillis drawing up the os calcis." "There is occasionally a natural depression between the occipital and parietal bones. Surgeons have been deceived by this variation and have trephined to their sorrow." Most of the occipital bone is "objectional to the trephine." Keats apparently found this part of surgery dull, for he has drawn pictures of flowers and fruit at the side of these notes. Elsewhere is a little drawing of a foot.

The first lecture on physiology gives a classification of the chief functions of the body. The second treats of the blood; in it we are told that the heat of the blood is supposed to be from  $98^{\circ}$  to  $100^{\circ}$ , that Mr. C., by placing thermometers in the left ventricle and right auricle of a dog, showed that venous and arterial blood had the same temperature. Fibrin is so called from being composed of fibrous particles. The various theories as to the coagulation of the blood are shown to be unsound. Mr. C.'s opinion is that it is prevented from coagulating in the body by nervous energy; it does not coagulate in an animal killed by lightning. Lecture 3 describes the arteries. The function of the elastic coat as an aid to circulation is emphasised; there is every probability that a muscular coat exists; arteries have vasa vasorum, absorbents and nerves. The red particles of the blood are a recent discovery; they appear to be bladders not completely full; they have the power of changing their figure; their size is supposed by Dr. Woollaston to be  $\frac{1}{4000}$  part of an inch. Lecture 4 treats of arteries and their diseases. When blood is thrown into an artery it elongates and convolutes. The slowest pulse Mr. C. heard of was seen by Mr. Stocker and Dr. Cholmeley, it was in general twenty, and sometimes twenty-eight or as low as fourteen. In typhus, stimulants will reduce the pulse. Digitalis considerably diminishes the frequency and pace of the pulse. Ossification of arteries is a constant effect of age, the young who lead a life of intemperance are subject to ossification. Those who have been much addicted to study, from keeping up a continual determination of blood to the brain, have often the vessels of that part ossified. Lecture 5 is headed, "Diseases of Veins." We are told how to treat varicose veins. "It has been practised to cure ulcers by tying the vein, but as this is

extremely dangerous it is now disused." A good deal is said about aneurysmal varix and its treatment, for in those days this sometimes followed venesection. Mr. J. Hunter tied the biliary duct of a dog; the absorbent vessels of the liver were found to be gorged with bile. Mr. Cline was going to operate for cataract; the point of the knife (which accidentally had broken off and remained in the cornea) was absorbed. Dr. Marcet's experiments on putrefaction are mentioned. The composition of chyle is given. Lecture 6 treats of the absorbent glands; much of it has to do with medical treatment. "People of warm climates have very large absorbents; in them the thoracic duct is twice or three times the size of that of an European." Lecture 7 is headed, "Reticular Membranes." Acupuncture is described as the treatment for œdema. Lectures 8 (9 absent) and 10 treat of the nervous system. The spinal marrow is composed of two columns, of late it has been discovered that these at the upper part cross each other. The first office of the nervous system is that of sensation. "Experiments have shown that the division of the par vagum on both sides is followed by destruction of life, but, if divided separately at intervals of time, the animal may live, showing satisfactorily that the nerve recovers its function after reunion." Mr. C.'s opinions are given; from Keats' wording he probably had not got them correctly, but when he notes that "In disease medical men guess, if they cannot ascertain a disease they call it nervous," we can imagine the lecturer to be accurately reported. Mr. C. had seen a dissection of the nerves in a man who had *tic douloureux*. The last entry about the nervous system is, "The present opinion therefore is that a fluid, like that of the electric, is excreted by the brain, which is thence communicated along the nerves."

The eleventh lecture treats of muscle and tendon. It is supposed that muscular fibre is hollow. In the last lecture there is more about muscle: "If a man throw his muscles into action while in water it will not rise in the vessel." Next cellular membranes are described: "Cancer is an inflammation which is common at first, but having a peculiar species of excretions, depending upon the state of the constitution." "In removing these the smallest portions must not be left." Glands are divided into three kinds: 1st, as the pleura, peritoneum, mucous membrane, etc.; 2nd, follicles or pores, as in the tongue, tonsil; 3rd, large glands having an excretory duct, such as the liver, kidney, etc. In secretion a gland performs a function similar to that by which cream is changed into butter and butter-milk. Nerves are supposed to control this operation.

It is a severe test to judge any lectures by a student's notes, but the impression formed by reading these is that they were interesting and likely to fix the attention of the student. In all of them there are many allusions indicating the bearing of physiology on surgery. They well represent the knowledge of the day; we must remember that at the time they were given microscopy was in its very infancy, that reflex action was unknown—it is in these lectures ascribed to sympathy, and that modern physiology was distant.

The question of who was the lecturer is interesting. In the anatomical notes Mr. C. is mentioned six times, in the physiological part fourteen times, in sentences beginning "Mr. C. believes," or "Mr. C. has seen," or with similar words, so that it is certain that Mr. C. was the lecturer. In the *Medical and Physical Journal* (xxxii, 258, 1814, and xxxiii, 75, 1815) we are told that, at the combined Medical Schools of Guy's and St. Thomas' "Lectures on Anatomy and the Operations of Surgery will be given by Mr. Astley Cooper and Mr. Henry Cline, and on the Principles and Practice of Surgery by Mr. Astley Cooper." In vol. xxxiv (1815), vol. xxxv (1816) and vol. xxxvi (1816) the same notice appears with the additional information that "Dr. Haighton will lecture on Physiology or the Laws of Animal Economy." During these years, the students of Guy's and St. Thomas's attended the lectures in both Hospitals. Mr. Astley Cooper and Mr. Henry Cline, junior, lectured at St. Thomas', Dr. Haighton at Guy's. The only other lecturers whose names began with C, were Drs. Curry and Cholmeley, both of whom lectured on Medicine. It is clear, therefore, that Mr. C., who lectured to Keats, might have been either Mr. Astley Cooper or Mr. Cline. In Feltoe's *Memorials of John Flint South*, 1884, we read (p. 33): "The first twelve lectures given by Astley Cooper we were accustomed to call the physiological lectures—on what, however, has long since been called by the French general anatomy—in which a slight sketch is given of the several structures of the body and the functions they were destined to be engaged in." Keats makes notes of a course of twelve lectures and heads them Physiology, so that these notes were almost certainly taken from Astley Cooper's lectures. South says, "These lectures were followed by about ten on the bones, four of which were devoted to those of the head and face, also given by Cooper." "Thus much of the Anatomical course having been gone through, Mr. Henry Cline commenced his portion of it." "Cline's lectures commenced with the muscles of the head." Now Keats' anatomical notes are entirely concerned with bones and ligaments, including

those of the head and face, except that there are short notes on the contents of the thorax and a very brief mention of vessels. There is nothing about the anatomy of muscles. So the evidence is strong that his anatomical notes also are from lectures by Astley Cooper, although, as we have already seen, they are so neat that they were probably written out after the lecture, but as the physiology notes are sometimes written around the anatomical, Keats attended the anatomical lectures before the physiological. South's statement that the first twelve lectures were physiological refers to 1813; it is quite likely that, two years later, when Keats attended, Astley Cooper gave the anatomical first, for this would be the more natural order. Another reason for believing the lecturer, Mr. C., was Astley Cooper is that he was far more popular of the two. South (*loc. cit.* p. 32) tells us: "Almost to the minute he (Astley Cooper) was in the theatre, where loud and continued greetings most truly declared the affectionate regard his pupils had for him. His clear silvery voice and cheery conversational manner soon exhausted the conventional hour devoted to the lecture; and all who heard him hung with silent attention on his words, the only sounds which broke the quiet being the subdued pen-scratching of the note-takers." (This reminds us of Keats and his note-taking.) Henry Cline, junior, who was lecturing at this time with Cooper, was a very different kind of person. Again quoting South: "He was a tall, sickly and very plain man, marked with smallpox and very shy . . . he went on in a quiet monotonous tone and very slowly . . . we never got out under an hour and a half and sometimes even three-quarters, and . . . very tiresome work I found it, as did my fellow-students, who never filled the theatre as they flocked to it when Cooper lectured." We can well believe that Keats did not attend Cline's lectures.

Astley Cooper's lectures on Surgery were published in 1824. The matter is quite different from that of Keats' Notes; nevertheless, we find so many similarities that Cooper must have given, not only the lectures on surgery but also those on anatomy and physiology, to which the poet listened. For example, in both the lectures on Surgery and Keats' Notes we find (*Surgery*, vol. i. p. 90, Notes on Lecture 6), "friction and movement of joints, as advised by Mr. Grosvenor of Oxford, highly recommended," also (*Surgery*, vol. iii. p. 262) we read, "Koschiusko,\* the Polish General, had his sciatic nerve injured by a pike." In the Notes (Lecture 10), "The patient K. . . . having had the sciatic nerve divided by a pike wound." In

\* Keats published a sonnet on him. *Examiner*, February 16, 1817.

both *Surgery*, vol. ii. p. 43, and Notes, Lecture 4, two examples of aneurysm produced by exertion are given; it is stated that an aneurysm may be produced by dilatation of the whole circumference of an artery and spurious aneurysms are mentioned. In *Surgery*, vol. i. p. 180, and vol. iii. p. 261, and in the Notes, Lecture 10, the same experiments are given to prove that nerves unite after division. Further, there are many instances in which the teaching in the Surgical Lectures and in the Notes is so similar, that, taken together they form strong proof that the lecturer is the same: see, for example, avoidance of wounding the intercostal artery, wounds of the bladder, fractured neck of the femur, aneurysmal varix, tying of veins. The evidence appears complete that Keats' note-book contains notes, made entirely by him, of lectures by Sir Astley Cooper.

When in October 1815, at the age of nineteen years and eleven months, John Keats entered as a medical student at Guy's Hospital, externally the building looked much the same as to-day, except that only the part built with Guy's money was then erected, the hospital being bounded at the back by Collingwood Street running east and west; this street has now entirely gone, being covered by extensions of the hospital.

The front entrance of St. Thomas's Hospital was in the Borough High Street, the building extended back, eastwards, towards Joiner Street, and the south side of the most eastern part of it faced into St. Thomas's Street opposite the main entrance to Guy's. Until 1814 Astley Cooper and Henry Cline, junior, had lectured in the old, inconvenient theatre at St. Thomas's, which in that year was replaced by one more commodious, where Keats, at two o'clock in the day, during the winter 1815-16, heard the lectures to which his notes refer. It formed part of the new anatomical buildings, containing a central hall, museum, dissecting room and lecture theatre, which, says South, "was one of the most handsome and best fitted rooms for seeing I have ever seen." There was seating accommodation for 290. Astley Cooper and Henry Cline each gave £1000 towards the expense of this new building. A few students of the "United Hospitals" of Guy's and St. Thomas's dissected in a small dissecting room at Guy's, but the great majority did this part of their work in the new room at St. Thomas's, so most likely Keats dissected there, and, as the *London Dissector* was the popular book of the day, he probably used this to guide him.

We can picture his clinical work from what we know of the teaching at the time. He had to accompany Mr. Lucas, junior, whose dresser he was, on his surgical rounds, carrying a tin

plaster box, in shape not unlike a table-knife box. Possession of such a box was a dresser's wand of office, and usually he was very proud of it. Often the surgeon was a little late, the students passing the waiting time in horse-play. The crowd of students walking the wards varied with the surgeon; if he was popular only a few could hear his remarks or see the patient, but most of the surgeons did little bedside teaching. When the patient was first seen, the surgeon either did, or superintended, the dressing and bandaging; if the dresser was trustworthy, the subsequent treatment was left much in his hands. The more diligent took notes, but this was not compulsory. Urgent cases were admitted at the discretion of the dresser; each took a week's duty, during which he had to live in the hospital at his own expense. If he considered it necessary, he could send for his surgeon. The most dramatic part of a student's life was to attend operations, a horrible and unnecessary procedure, for, very few would, in after life, dare to perform the operations they saw done in the theatre. Guy's had one large operating theatre, St. Thomas's two smaller. This is South's account of what happened: "The pupils, packed like herrings in a barrel, but not so quiet, as those behind were continually pressing on those before, often so severely that several could not bear the pressure, and were continually struggling to relieve themselves of it, and had not infrequently to (be) got out exhausted. There was also a continual calling out of 'Heads, heads' to those about the table whose heads interfered with the sight-seers, with various appellatives, in a small way resembling the calls at the Sheldonian Theatre during Commemoration. . . . I have often known even the floor so crowded that the surgeon could not operate till it had been partially cleared." After the operations at St. Thomas's were over the students tore across the street to be in time for those at Guy's, and again the same disgusting struggle for places took place. "So long as the patient did not make much noise I got on very well, but if the cries were great, and especially if they came from a child, I was quickly upset, had to leave the theatre, and not infrequently fainted . . . the atmosphere almost stifling." What must have been the feelings of the patient walking into a crammed theatre like this; what must have been the feelings of Keats with his nature, attending such a loathsome, cruel exhibition!

The three surgeons at Guy's during Keats' studentship were Thompson Forster, William Lucas, junior, and Astley Cooper. The first, who had been in the army, was a very gentlemanly old man, with the upright gait and carriage and spotless neat-

ness of an old soldier. He did not operate frequently. Mr. Lucas, junior, to whom Keats was dresser, was a "tall, ungainly, awkward man, with stooping shoulders and a shuffling gait, as deaf as a post, not overburdened with brains of any kind, but very good-natured and easy, and liked by everyone. His surgical acquirements were very small, his operations generally very badly performed and accompanied by much bungling, if not worse. He was a poor anatomist and not a very good diagnoser, which now and then led him into ugly scrapes." It is said that after Astley Cooper's appointment Lucas and Forster rarely operated unless he was present. It is not likely that either of the two seniors could have had an influence on Keats, but Astley Cooper was very different. Tuesdays and Fridays were the days when he went round, and, as his carriage drove up quickly, he was out in a moment, skipping up the steps like a bright schoolboy, surrounded by a crowd of pupils; rushing up the staircase, pupils pushing and scrambling to get near, he went round the wards, the audience hanging on every word he said. His visit only lasted about half or three-quarters of an hour, and he then went quickly across the street to lecture on anatomy at St. Thomas's. His surgical lectures were given in the evening. The students crowded round the door directly it was opened, rushed to secure the best places; by the time Astley Cooper arrived they were sitting on the passages and stairs, fully three hundred being in the theatre. He was a remarkably handsome man, universally regarded as being at the head of his profession, a great teacher, an investigator, a bold and brilliant surgeon, a true and honest friend, always willing to help students and doctors, and with a wonderful gift of remembering them and all about them even if he had not seen them for years. It is greatly to his credit that he noticed and took an interest in "little Keats," so called by his fellow-students, as he was only just over five feet high. That he did so we know from this sentence by South: "George Cooper told me that whilst at Guy's Hospital, where he was dresser to Astley Cooper for eighteen months, he lived in St. Thomas's Street, at a tallow-chandler's named Markham, where John Keats, the poet, lived with him, having been placed under his charge by Astley Cooper." Possibly the great surgeon was attracted by the poet's dark-brown brilliant eyes and golden-red hair.

The physicians to Guy's in Keats' time were James Curry, Alexander Marcet and James Cholmeley. The assistant physician was James Laird. Under which of them Keats worked is not known. Curry, a small, thin man with fine

features and great intelligence, was irascible, peevish and overbearing. He was not liked, and everyone feared to come near him. Marcet was a distinguished chemist, mostly occupied with chemical investigations. Cholmeley appears to have been a good practical physician, and of Laird we know nothing, except that poor health compelled him to retire from the hospital staff in 1824. It is unlikely that any of these had much influence on the poet.

During the winter session of 1815-16 lectures on the Practice of Medicine were given by Drs. Babbington and Curry, on Chemistry by Drs. Babbington and Marcet and Mr. Allen, on Experimental Philosophy by Mr. Allen, on the Theory of Medicine and Materia Medica by Drs. Curry and Cholmeley, on Midwifery and Diseases of Women and Children by Dr. Haighton, on Physiology and the Laws of Animal Economy by Dr. Haighton, and on the Structure and Diseases of the Teeth by Dr. Fox. Printed notes of the Lectures on Medicine still exist; they occupy over 200 pages. In 1802 a course by Dr. Curry on Pathology, Therapeutics and Materia Medica consisted of seventy lectures. All these lectures were given at Guy's in the theatre, which stood unaltered until a few years ago; it was always known as the old Chemical Theatre.\* We do not know how many of all these lectures in this theatre Keats heard, but he must have listened to some, for, before he could be admitted to his final examination at the Apothecaries' Society, he had to produce certificates to show that he had attended two courses of lectures on Anatomy and Physiology, two on the theory and practice of Medicine, two on Chemistry and one on Materia Medica. This last he would naturally have done at Guy's, not at the Apothecaries' Society as has been stated. Babbington had retired from the post of physician in 1811; therefore it is unlikely that Keats, even if he listened to him, knew him personally, which is a pity, for in addition to his high professional attainments his personality was such that he was beloved by everybody and was one of the most delightful of men; but the young poet almost certainly listened to Curry for "he was one of the most fluent and attractive lecturers I have ever known; his language was unexceptionable, and his words flowed in an unbroken torrent. The attention of his large class never flagged."

Keats lived alone in 8, Dean Street, close to Guy's, for about three months from October 1815. He wrote to Clarke: "Although the Borough is a beastly place in dirt, turnings and

\* There is a picture of this in the *History of Guy's Hospital* by Wilks and Bettany.



windings, yet No. 8, Dean Street, is not difficult to find; and if you would run the gauntlet over London Bridge, take the first turning to the right,\* and, moreover, knock at my door, which is nearly opposite a meeting, you would do me a charity, which, as St. Paul saith, is the father of all virtues." Leaving Dean Street, in December 1815 or January 1816, he went to St. Thomas's Street, two fellow-students living with him and Cooper; these were Henry Stephens and George Wilson Mackereth. We know from the dates of some of his poems that while living in St. Thomas's Street he was occupied with poetry. For example, as a valentine on February 14, 1816, he wrote for his future sister-in-law: "Hadst thou liv'd in days of old." Also about the same time he wrote "Calidore." His first published poem, the sonnet, "O Solitude," appeared in the *Examiner* on May 5, 1816. The impressions of summer strolls in his poetry of the next few months strongly suggest that during them he paid visits to Leigh Hunt at Hampstead, while the lines,

" O Solitude ! if I must with thee dwell,  
Let it not be among the jumbled heap  
Of murky dwellings,"

and

" To one who has been long in city pent,"

indicate that he was irked by the peculiarly close and disagreeable surroundings of the Borough, worse then than now, for the large clearings necessitated by the railway had not been made. He can hardly have been living at Hampstead during the summer of 1816, for he had to attend to his lectures and clinical work and his examination in July was in front of him. Means of communication were then so tedious that medical students had to live near the hospital. He almost certainly lived in St. Thomas's Street till after his qualifying examination at the end of July 1816; he then went to Margate for a holiday and returned to London in September. Whether he went back to his old lodgings in St. Thomas's Street is uncertain, probably he lived with his brother in the Poultry, whence he could easily walk to Guy's, where he had not finished his appointment to Mr. Lucas, which, as we have seen by the

\* Keats makes a slip. Coming over London Bridge in 1815, the first turning to the right was Pepper Alley, but, if you turned to the left, the first turning was Tooley Street, south of which, where London Bridge railway stations now stand, was a collection of small streets forming the district known as Berghene or Petty Burgundy. One of these running south was Dean Street, the southern end of it was a little east of Guy's. This end, which was not abolished by the railway, has always been a favourite lodging for Guy's students. The Guy's surgeon, Mr. Edward Cock, had consulting rooms there for many years until his retirement. It is now known as Stainer Street.

entries in the Medical School Office books, he took for the second time in March 1816 for twelve months. But there is no direct evidence of his having worked at Guy's after his qualification. His guardian and trustee, Abbey, wished him to settle in Tottenham or Edmonton as a surgeon.\* Keats replied that he would not, and that he intended to rely on his ability as a poet. He never practised, but he refers, in several of his letters,† to a slender possibility that he might.

The Apothecaries' Society, in 1815, obtained the right to grant a qualification; the regulations required a knowledge of Latin, attendance on lectures as just mentioned, six months' attendance at the practice of a hospital and an apprenticeship of five years to an apothecary. This last Keats had not fulfilled, having been apprentice to Mr. Thomas Hammond for only four years. How this difficulty was overcome we do not know, but he presented himself for examination on July 25, 1816. He passed with ease and credit and thus became qualified. It used to be stated that he did not qualify, but the later publications about Keats have shown that this is an error. It is therefore of interest that I have discovered the list of successful candidates. It is published in *The London Medical Repository* (vi. 345, 1816); it contains seventy-one names, and the forty-ninth is John Keats, Wilston, Edmonton. The list is not alphabetical; presumably the order is that in which the candidates presented themselves. Mr. A. B. Watson, clerk to the Society, tells me that Wilston would be the name of Hammond's house at Edmonton. In the Minute Book of the Court of Examiners of the Apothecaries' Society under the date of July 25, 1816, we read: "John Keats was examined by Mr. Brande and the Court granted him the Certificate for which he had applied." This examiner was Everard Brande, brother of William Thomas Brande, the famous chemist.

During his medical studentship there were two Keats in the same body. The one was, as is shown by his notes, by the rapidity with which he qualified and by the ease with which he passed his examination, a diligent medical student, sufficiently outstanding to attract the attention of the great Sir Astley Cooper; the other was the poet. The last at times thrust the medical student aside. We have seen that during his studentship he wrote and published poetry; we know, from his own confession, that occasionally at lecture "there came a sunbeam into the room, and with it a whole troop of creatures floating in the ray, and I was off with them to Oberon and fairyland."

\* Amy Lowell, *John Keats*, vol. i. p. 184.

† Buxton Forman, *John Keats* [Gowans and Grey], 1901, Index, vol. v. p. 244.

He would sit so often in the window-seat of the St. Thomas's Street lodgings that it was called Keats' place. The story is that here, dreamily resting one evening, while Stephens was studying medicine, the young poet broke out with the announcement he had composed a new line : \*

"A thing of beauty is a constant joy."

"What think you of that, Stephens?"

"It has the true ring, but is wanting in some way," says Stephens and goes on reading. An interval of silence and then Keats,

"A thing of beauty is a joy for ever,"

and thus was born in St. Thomas's Street one of the imperishable lines of English Poetry.

\* *The Asclepiad*, vol. i. p. 149, 1884.

# WEIGHT, VITAL CAPACITY, PULSE RATE BEFORE AND AFTER EXERCISE, AND PHYSICAL FITNESS IN HEALTH

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IN comparing the results of fractional test-meal and x-ray examinations of the stomach with the physical fitness of healthy students, the opportunity was taken to carry out a series of tests for physical fitness and to compare the grading by these with that arrived at more directly by the man's physique and athletic performances. The gastric investigations have already been described in this Journal,<sup>16, 17</sup> and those who took part in both series of tests have been denoted by the same numbers to make reference easy.

The subjects examined were eighty students at Guy's Hospital, the age of the majority being between 18 and 25. Absolute accuracy cannot be claimed for the observations, because many of them were made by the students themselves, but either Mr. R. R. Traill or myself was always present to see that they followed the prescribed routine, and frequently checked the measurements. The men were not selected in any way; some were good athletes, some took no exercise of any sort.

## PART I. METHODS AND TESTS USED

The following measurements were made in all cases: (1) Body weight, Circumference of chest, and Stem length. (2) Vital capacity. (3) Pulse rate at rest. (4) Pulse rate after a fixed exercise.

*Body weight.*—The men were weighed in their clothes and one-twentieth of the gross weight was deducted to give the body weight naked. This figure was arrived at by weighing six men, selected at random, with and without clothes. The heaviest clothes were less than 1/15th, and the lightest were more than 1/25th of the gross body weight. Calculated weight was taken from Dreyer's tables<sup>12</sup> as the average of the two values obtained from the chest and from the stem length. Calculated vital capacity was obtained in the same way. The

\* The experimental part of this work was done during the tenure of the Hilda and Ronald Poulton Fellowship at Guy's Hospital.

chest and stem length<sup>11</sup> were only measured to the nearest centimetre, as more detailed measurement gives a false sense of accuracy. That these assumptions do not lead to any gross error is shown by a comparison of these results with those obtained recently by Hobson,<sup>15</sup> working in Dreyer's laboratory (see Table I).

*Vital capacity.*—This was measured standing and each subject made three attempts, the highest reading being recorded; some gave the highest value the first time, but most men improved with practice, and the difference was often nearly 10 per cent. The men were allowed to see the dial, as the competition produced certainly led to higher readings.

TABLE I.

PERCENTAGE DIFFERENCE OF OBSERVED AND CALCULATED WEIGHT AND VITAL CAPACITY IN NORMAL STUDENTS.

	Observer.	No. of cases.	Above +15%.	+10 to +15.	+5 to +10.	-5 to +5.	-10 to -5.	-15 to -10.	Below -15%.
Weight . . .	J.M.H.C.	80	1	0	5	38	27	9	0
Weight . . .	F.G.H.	46	0	0	1	23	12	8	2
Vital capacity .	J.M.H.C.	80	5	5	12	28	16	12	2
Vital capacity .	F.G.H.	46	8	2	4	21	5	3	3

*Pulse rate at rest.*—It was not possible to get this under absolutely standard conditions, but generally it was counted at about 11. a.m. No one had taken any exercise within two hours of examination, some men had been sitting reading, and others had been engaged in laboratory work. The men rested for five minutes, and if the pulse had then reached its usual rate, this was considered sufficient; if not, or if it still appeared high, they rested for a further ten minutes. Probably the pulse rate at complete rest is lower than these figures.

*Pulse rate after exercise.*—The importance of this was emphasised by Pembrey, Cook and Todd<sup>6, 9</sup> and for women by Hartwell and Tweedy,<sup>10</sup> and the following method was described in detail by Hunt and Pembrey.<sup>13</sup> The same exercise was used in every case, stepping on and off a wooden block 13 inches high twenty-eight times a minute for three minutes. An interval of five seconds was allowed for the subject to sit down, and the pulse was then taken for two minutes. The four half-minutes were separately recorded and in the fourth half-minute the pulse was often nearly back to its normal rate in the fit men. The "pulse ratio" was found by dividing the number

of heart-beats in the two minutes following the exercise by the number at rest. This rate was chosen because by trial it was found convenient for most men. One minute's exercise did not generally produce enough effect, and three minutes' generally made a man just out of breath, and sometimes caused slight sweating, as the subject was not allowed to discard any clothes.

Before the results can be discussed the classification adopted to divide the students into four groups will be described. Various games secretaries, teachers and fellow-students who knew nothing of the results of the tests were asked to group the men, and on the whole the results agreed well. Seventeen were classed consistently as athletes or above the average and were put in group A. Most of these were playing games regularly for a good hospital side. Nineteen were generally classed as above the average, and were put into group B1. They were nearly all playing games, but less regularly and for less successful sides. Twenty-three were generally classed as average, though often one opinion "above average" cancelled one "below average"; these were included in group B2. As none of these were playing games regularly and most of them not at all, the word "average" was used in a rather unfavourable sense. Twenty-one were included in C, the most heterogeneous group, because it contained eleven men, classed sometimes average and sometimes below average, who differed very little from B2; and ten classed consistently as below average or unfit. The first eleven men were put in C instead of B2 to make the groups of almost equal size for comparison, though this tends to minimise the difference between group averages. This is a rough method and I was surprised that it gave as good agreement as it did. Judging by the athletic record of the men (see Table X) this appeared to be the deciding factor.

Group A is referred to as "athletes," B1 as "above average," B2 as "average," and C as "below average." If B2 represents the average medical student, the number taking regular exercise is lamentably small. No doubt the medical curriculum is crowded, and the amount of work to be done very large. But a man who takes no vigorous exercise at this age can hardly have grasped one important physiological truth, that health cannot be maintained without an equal development of all sides of the body; nor is he likely to prove a good guide in the many minor disorders arising from neglect of this rule.

The average age, weight, stem length and chest measurement of the four groups are very similar, and the small changes

cannot be responsible for the differences obtained between the four groups.

## PART II. DISCUSSION OF RESULTS FOR EACH GROUP

*Relation of observed and calculated body weight.*—This appears to be the most valuable part of Dreyer's book <sup>1</sup> and in more than 85 per cent. of men examined the weight lies within 10 per cent. of its calculated value (see Table I). Only one is above this and nine below, and none are more than 15 per cent. below. There is little difference between the groups, but the athletes are slightly heavier than the others, and as there is little change between the stem length of the four groups, this must depend on the increase in the size of the chest and in muscular development.

None of the athletes are 10 per cent. below calculated weight, though some are nearly this. The nine men fall equally between the other three groups. Five of them would have been described as thin by the most casual observer and one as very tall and thin. It is not quite clear if Dreyer means by abnormal, diseased or merely unusual. The latter is obviously true, the former is almost certainly not. Of the nine men who were 10 per cent. below Dreyer's standard for their weight, five were fit and showed no other abnormality, and four were unfit or at least showed other abnormalities.

Deviations of less than 10 per cent. do not appear to be of any importance; deviations of more than 10 per cent. are generally obvious to the observer but difficult to estimate accurately without some such method.

Considering the enormous variations often found between the weight calculated from the chest and from the stem length, the agreement of the average is the more striking, *e.g.* No. 34, where the weight from the stem length was 71.5 and from the chest 51.1, giving an average calculated value of 61.3, which agreed closely with the weight actually found, *viz.* 59.6. One serious drawback to this method is that the man who is too fat may appear to be of standard weight, because the chest measurement is much too large owing to subcutaneous fat. In such cases the weight calculated from the stem length only gives a much truer picture, *e.g.* No. 66, who was certainly very fat, was 5 per cent. below weight calculated in the usual way, but 24 per cent. above calculated from the stem length. The true state of affairs is between these two, considerably nearer to the latter. The same criticism does not apply with equal force to the man below weight,

because loss of subcutaneous fat cannot diminish the chest measurement to the same extent that excess may increase it.

*Relation of observed and calculated vital capacity.*—As would be expected, there is not such close agreement for vital capacity as for weight, because the latter is a purely anatomical measurement and the former depends on structural and functional factors. The general range is given in Table II, which shows

TABLE II.

DISTRIBUTION OF VITAL CAPACITY IN DIFFERENT GROUPS.

(Expressed as percentage deviation from the calculated vital capacity.)

	More than + 10%.	+ 10% to - 10%.	Less than - 10%.
Athletes . . .	4	13	0*
Above average . .	3	15	1*
Average . . .	2	18	3*
Below average . .	1	10	10

\* These figures should perhaps read 2, 3, and 4 as five men improved their vital capacity to the class above at a second attempt (see text).

well that in the majority of subjects (70 per cent.) the vital capacity is within 10 per cent. of its theoretical value. A vital capacity 10 per cent. below the standard is evidence of unfitness, but one 10 per cent. above is not much evidence of fitness, for of the ten men with high vital capacity, five were certainly very fit, but three were unfit or abnormal in some other way.

In Table II fourteen men are shown with a vital capacity 10 per cent. below standard, of whom four were classed as average or above and ten as below average. But at the first attempt nineteen failed to reach this standard. Unluckily they were not all given a second attempt, but of the seven who were, two athletes and three men who were average or above raised their vital capacity to within normal limits when they were told they had done badly the first time and were given a second chance, while the two who were below the average were unable to do so.

Is the vital capacity constant at different times? Twenty men were tested again six months later without any special comment about their previous attempt, and in seven the difference was less than 200 c.c., in eight there was a change of 5 per cent. or less, and in five a change of more than this, generally about 10 per cent. But taking them as a whole some were better and some were worse, the average improvement being very small.



Nine others (including those already referred to) were re-examined, and before the second attempt they were told that the first had been below the standard expected of them. In all but one there was an improvement, and the change was generally enough to move the man from the unfit to the average group, the mean increase being 5 per cent.

As most of the men on the first occasion were keen to beat others who were present, 5 per cent. must represent the minimum change which can be brought about by a special effort. Such a fact would go far to explain the diminished vital capacity which was found in flying officers who were tired out and unfit for further flying.

Supposing that 5 per cent. represents the usual increase with a special effort, one cannot be sure of any real abnormality unless the vital capacity is 15 per cent. below standard. This would include only four men, two of whom (36 and 41) improved at a second examination and two of whom (63 and 77) were included as unfit on other grounds. Of eighteen who were 10 per cent. below Dreyer's standard at a first attempt, only nine were classed as unfit on other grounds. All calculations have been done for Dreyer's Class A because the use of the other classes is to some extent prejudging the question about which information is desired.

*Pulse rate at rest.*—Even under these conditions, when the minimum had sometimes not been reached, a pulse rate over 78 rarely occurred in men who were really fit. Wider variations have been given,<sup>6, 14</sup> but it is doubtful if the more rapid can be regarded as normal in young men.

The influence of weight on the pulse rate was emphasised by other workers in this laboratory,<sup>14</sup> and the following table shows it very clearly.

TABLE III.  
INFLUENCE OF WEIGHT ON THE PULSE RATE AT REST.

Weight of body in kilos.	Number of subjects.	Pulse rate.		
		Average.	Extreme range.	Usual range.
45-55	4	76.5	68-86	76-86*
55-60	17	73.3	56-90	84-90*
60-65	20	70.0	58-84	58-80*
65-70	26	68.5	57-82	57-82
70-75	12	64.7	48-74	55-74*
75-85	5	63.4†	48-78	48-71*

\* Excluding one subject only.

† If one unfit man was excluded the average rate was just under 60.

The average pulse rate and its range both get slower as the weight increases, but there are considerable individual variations. If the weight is over 70 kgms. (11 stone) the pulse is generally under 70, and sometimes under 60; if the weight is under 60 kgms. (9½ stone) the pulse is rarely under 60 and generally over 70. For weights between 60 and 70 kgms. it is more difficult to make any definite statement.

The pulse rate at rest is certainly the easiest and possibly the most useful of these observations, and a rate above 78 with average weight is a valuable sign of unfitness. A slow pulse rate cannot be taken as conclusive evidence of fitness because weight is an additional factor, but it gives an important indication. The average pulse rate and its distribution among the different groups is shown in Table IV.

TABLE IV.  
PULSE RATE AT REST AMONG THE DIFFERENT GROUPS.

	Below 65.	65 to 70.	71 to 76.	78 and above.	Average rate.
Athletes . . .	9	4	4	0	64.8
Above average . . .	4	7	7	1	69.0
Average . . .	6	7	5	5	70.3
Below average . . .	2	6	4	9	74.5

*Pulse rate in various animals.*—Before considering the pulse rate after exercise it is interesting to compare the influence of weight in man and in various animals. Fig. 1 shows that the fall in pulse rate with increasing weight is applicable to nearly all birds and mammals. The curve for the short range which is covered by these subjects is similar to the curve for the fall in the pulse rate from birth to adult life taken from Tigerstedt.<sup>4</sup> The latter is rather higher, and this may well be due to the inclusion of girls and women, for Guy<sup>5</sup> has shown that a woman's pulse is on the average ten beats faster than a man's, and that difference can be seen at any age after seven.

Both these curves agree well with the curve obtained for warm-blooded animals over a very much wider range. The figures on which it is based are mostly taken from Buchanan,<sup>8</sup> and for the smaller animals the rates were counted by the electrocardiogram. They are given in Table XI at the end. All the available animals have been used for average figures to draw the curve, except the hare and the pigeon, which have unusually slow pulse rates for their size, possibly because of

the prolonged effort these animals are accustomed to make. This is interesting in view of the slower pulse of the athlete.

No doubt the fall in pulse rate with increasing weight is part of the general law discovered by Rubner, that metabolism varies with the surface of the warm-blooded animal and so is relatively much greater with the smaller animals. For rapid metabolism a large pulmonary ventilation and a rapid circulation rate are helpful, and the pulse rate probably depends largely on this.

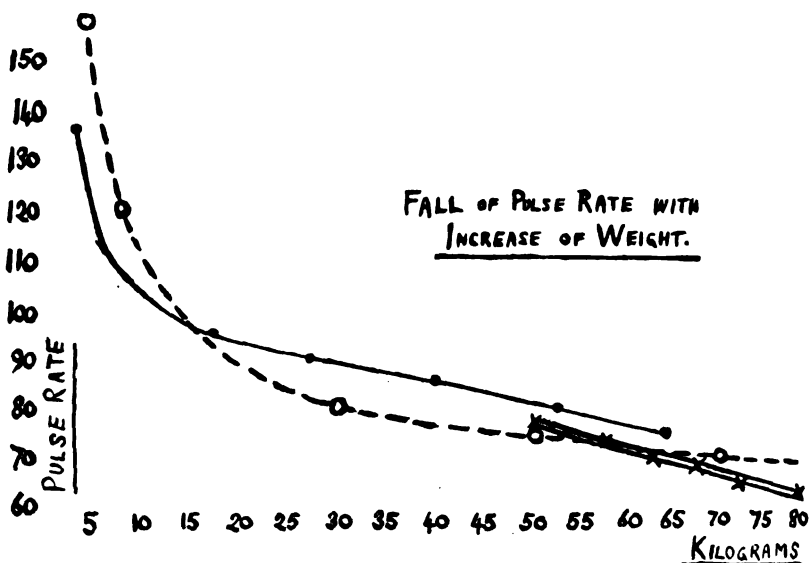


FIG. 1.—Fall of Pulse Rate with Increase of Weight.

— — — broken line is average curve for all birds and mammals given in Table XI (excluding the pigeon and hare).

— single unbroken line is curve for man from infancy to adult life taken from Tigerstedt.<sup>4</sup>

== double line is for the series of men considered in this paper. It is parallel to the curve taken from Tigerstedt, and is probably lower because it is compiled from men only and the other from men and women.

It seems reasonable that the rate of respiration should also vary in the same sense. Fewer data are available, but Table XI includes the respiration rate of some animals, for which there are records. In large animals such as the rhinoceros, lion and tiger it is between 6 and 10; in the monkey it is 19—not very different from man; while in a small animal like the rat it is 210. These observations, which are mostly taken from Paul Bert,<sup>3</sup> agree in general with what has been found for the pulse rate, but are less regular, probably because of the few data on which they based. The rate for the infant at birth and the boy at ten years taken from Quetelet<sup>1</sup>

agree moderately well with the rate for animals of similar size. I do not know any large number of figures to discover if weight is one of the factors influencing the very different respiration rates of adult man. A few individual cases seem to agree with it.

*Pulse rate after exercise.*—This was taken in each case for two minutes after the stepping exercise was finished and the values for the four separate half-minutes are given in Table X. There are very wide variations in the pulse ratio between individuals, from 2.4 to 3.8. The average and the distribution among the different groups are shown in Table V. As with the other tests, the unfit group is worse than the other three, which do not show much difference. More than half of the athletes and men above the average have a pulse ratio between 2.5 and 3.0. Nearly half of the average men have a pulse ratio between 3.0 and 3.5, and only one man of all these groups has a pulse ratio higher than this. On the other hand, of the men below the average nearly one-third have a pulse ratio above 3.5.

TABLE V.  
PULSE RATIO AMONG THE FOUR GROUPS.

	2.5 and below.	Above 2.5. Below 3.0.	3.0 to 3.5.	3.5 and above.	Average.
Athletes . . .	4	8	5	0	2.8
Above average . . .	3	11	5	0	2.7
Average . . .	2	9	10	2	2.9
Below average . . .	3	5	7	6	3.1

Obviously a really high pulse ratio is a reliable sign of unfitness, but the difficulty is with the low pulse ratio, which occurs almost equally in all the groups, because the high resting pulse rate of the unfit man gives him an artificial advantage by minimising the effect of the higher rate after exercise, *e.g.* in No. 78 the return of pulse to normal after exercise is extremely slow, but he has a pulse ratio of 2.7 because the resting pulse is 86.

To avoid this difficulty the average pulse rate for each half-minute after exercise has been plotted and a curve has been drawn to show the rate of fall of the pulse in six athletes, and in six men who were unfit (see Fig. 2). The difference in the first half-minute after work is greater than the difference in the resting pulse, but in the fourth half-minute after work it is even greater. The importance of the rate of return of

the pulse after exercise was emphasised by Pembrey, Cook and Todd,<sup>6, 9</sup> who pointed out the difference in trained and untrained men. It gives a more accurate and more detailed knowledge of the behaviour of the pulse and a better indication of a man's fitness than does the pulse ratio.

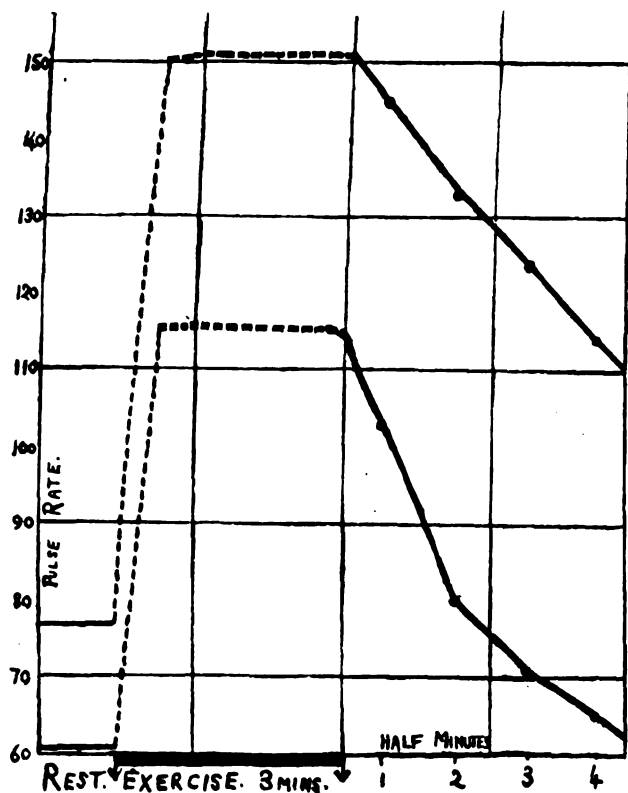


FIG. 2.—Pulse Rate Before and After Exercise.

(Three minutes, 28 steps a minute.)

Lower line shows average rate in six athletes. Upper line shows average rate in six men who were unfit. The rate after exercise is taken for the first four half-minutes (1, 2, 3, 4 on chart). The fit men have a slower pulse rate at rest and a more rapid return of the pulse rate to normal after exercise. The relative increase in the pulse rate immediately after exercise is almost the same in the two groups. The rate is shown rising steeply at the beginning of exercise as Buchanan found with the electro-cardiogram.<sup>7</sup>

In the unfit the pulse in the first half-minute after exercise was considerably higher than in the athletes, but it was also higher at rest, so that relatively the increase was about the same. If the pulse ratio was taken for the first half-minute after exercise only it would show no difference in the two groups, but it does show a difference because the fall after this does not

take place at the same rate, the difference being greatest in the second minute. Table VI shows the average pulse rate in the fourth half-minute after exercise and its distribution among the groups.

TABLE VI.  
PULSE RATE IN FOURTH HALF-MINUTE AFTER EXERCISE.

	Below 78.	78 to 90.	92 to 110.	Above 110.	Average pulse rate.
Athletes . . .	7	7	3	0	80.0
Above average . . .	9	7	3	0	80.0
Average . . .	7	6	8	2	88.0
Below average . . .	2	5	7	7	97.2

Even taking the pulse rate in the fourth half-minute after exercise, a glance at Table VI shows that though a high pulse rate indicates men who are below the average, and a low pulse rate generally excludes men who are below the average, it does not help in distinguishing between the athletes and the men who are just above the average.

As would be expected, all these four tests give better results for the athletes and men above the average. The greater the difference found between the groups the more likely the test is to prove valuable in individuals, and Table VII gives this comparison. For vital capacity the athletes were 2.5 per cent. above their calculated value and the men below the average were 5 per cent. below—a difference of nearly 8 per cent. The pulse ratio varied from 2.8 (athletes) to 3.1 (below average), a difference of 11 per cent. The pulse rate at rest varied from 64.8 to 74.5, a difference of 15 per cent., and the pulse rate in the fourth half-minute after exercise from 80 to 97.2, a difference of 22 per cent. This means that the pulse rate at rest and after exercise are more likely to be useful than the other two tests.

TABLE VII.  
COMPARISON OF TESTS ON THE FOUR GROUPS.

	Vital capacity.	Pulse rate at rest.	"Pulse ratio."	Pulse rate in fourth half-minute after exercise.
Athletes (A) . . .	102.5	64.8	2.8	80
Above average (B1) . . .	99	69.0	2.7	80
Average (B2) . . .	101	70.3	2.9	88
Below average (C) . . .	95	74.5	3.1	97.2
Percentage difference between A and C . . .	8	15	11	22

## PART III. DISCUSSION OF RESULTS FOR INDIVIDUALS

So far the results have been considered from the point of view of the groups of varying degrees of fitness, but the range of variation among the members of a group is so great that no one of these tests would be of much use in classifying them. For ordinary medical work the results for the individual are more important, and Table VIII shows all who could be picked out as abnormal by the various tests. It is difficult to draw a sharp line between fit and unfit and the result varies according to the dividing line chosen. For weight and vital capacity 10 per cent. below the calculated value has been selected, because Dreyer suggested that if either of these was 10 per cent. below, the man was probably abnormal. For pulse rate at rest 78 or above was chosen, because on looking through the tables this seemed to be the turning-point; possibly in another group of students a slightly different point would have been fixed on, but it would not have been very different. For the pulse ratio 3.5 or above was chosen because a lower figure, although it included a few men judged unfit on other grounds, included more who were judged quite fit. A pulse rate above 110 in the fourth half-minute after exercise is not an independent test, because naturally it is related to the rate at rest which has already been counted, but one may be high and the other relatively low, for of the nine men with a pulse rate above 110 in the fourth half-minute, only three had a pulse rate at rest of over 78 (Nos. 62, 67 and 79). The pulse ratio and the pulse rate in the fourth half-minute are even more closely related, but both are included in Table VIII because three men (62, 69 and 77) who had a pulse ratio below 3.5, but were judged unfit on other grounds, were picked out as abnormal by a high pulse rate in the fourth half-minute.

In Table VIII abnormalities in pulse ratio or in the pulse rate in the fourth half-minute after exercise are combined as one test. This gives four tests and there are 49 occasions on which a man was picked out as sufficiently below what was usual to be abnormal. None of these 49 bad results were given by an athlete; only five by men above the average, two of these by the same man, who was possibly not in good health; and only 13 by men grouped as average, three of these by the same man. On the other hand, 31 of the 49 were given by the 21 men who were classified as below average on other grounds. Of these, two men failed in three tests, nine others in two, and seven in one test only. Eighteen of the 21 men who were below the average failed in at least one of the four

TABLE VIII.  
MEN PICKED OUT AS ABNORMAL BY EACH TEST.

	Total number of subjects.	Serial numbers of men found abnormal by each test.					
		Athletes (1-17).	Above average (18-36).	Average (37-59).	Below average.		
					(60-66).	(67-73).	(74-80).
1. Weight more than 10 per cent. below calculated value . . .	9	—	18, 24*, 35	54, 58†, 59	62†	—	75†, 76*
2. Vital capacity more than 10 per cent. below calculated value . .	14 (+5) 15	(4) (14) —	19, (28), (36) 24*	(41), 43, 45, 58† 46, 49, 50, 55, 56	61, 63*, 66* 62†, 66*	68, 69*, 73 67*, 71, 72	74*, 75†, 77*, 78* 75†, 78*, 79*, 80
3. Pulse rate at rest 78 or above .	11	—	—	57, 58†	62†, 63*, 64	67*, 69*	74*, 76*, 77*, 79*
4. Pulse rate after exercise abnormally high from 4A or B . . .	9 8	— —	— —	57, 58 57, 58	62, 64 63, 64	67, 69 67	76, 77, 79 74, 76, 79
4A. Pulse rate in fourth half-minute after exercise above 110 . . .							
4B. "Pulse ratio" 3.5 or above .							

\* denotes men who were found to be abnormal in two tests (counting 4A and B as the same).

† denotes men who were found to be abnormal in three tests (counting 4A and B as the same).

The five men bracketed are not counted because they were able to raise their vital capacity to the normal standard at a second attempt, and there was no other evidence of unfitness. This is another drawback to the value of the vital capacity test.



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tests and 11 of the 21 failed in two or more. Table IX shows clearly that failure in one test is much more common among the men who are average and below average, and that failure in two tests is practically diagnostic of a man being below the average, for No. 24 was possibly not in good health and No. 58 might easily have been put in the lowest class. By using the tests combined in this way a much larger proportion of unfit men can be detected than by any single test, and practically no fit men are included with the unfit as they would be by the use of single tests.

TABLE IX.  
NUMBER OF MEN IN EACH GROUP FAILING IN VARIOUS TESTS.

	Failure in no test.	Failure in one test.	Failure in two or more tests.	Number in group.
Athletes . . . .	17	0	0	17
Above average . . . .	15	3	1	19
Average . . . .	12	10	1	23
Below average . . . .	3	7	11	21

Taking the men individually there are four who should not strictly be included as in good health, for 80 was sent to a sanatorium for suspected phthisis about ten weeks after he had been tested, and 38, 50 and 66 had all had rheumatic fever within the previous five years. Although 38 had acute heart block with his attack, he was passed as normal by every test and had made a good recovery, but 50 and 66 both had a pulse rate above 80 and had therefore probably not recovered completely. They were neither detected as abnormal by any other test except that 66 failed in his vital capacity; 80 was passed as normal by his weight, vital capacity and pulse rate after exercise and was only detected as abnormal by the pulse rate at rest. Of these four men known to have had serious diseases, three were picked out by a high pulse rate at rest, and only one by his vital capacity; none were picked out by any other test. No. 31 had a loud systolic bruit over the base of his heart, which had been present from birth, but recently he had been allowed to play games, and had done so with success and no symptoms. As there was no evidence of disease apart from the bruit, he probably had a patent foramen ovale. He succeeded in every test, and should be included as normal.

In addition to these men there are four others about whose good health I do not feel certain. All four (24, 62, 63 and 75) looked very thin and three were found to be 10 per cent. below

weight according to Dreyer's standards. Two were 10 per cent. below on their vital capacity. Three of the four had a resting pulse rate of 78 or above, and two had an abnormally rapid pulse after exercise. All four were picked out as abnormal by at least two independent tests, and two of them by three independent tests. The only other man who was picked out by three independent tests (58) was, in my opinion, below the average, though just included by others in the average group. A further proof that these men were unusual is that of the three who were examined by the fractional test-meal and with x-rays all were found differing from the normal in some way.<sup>16, 17</sup> No. 24 had normal test-meal findings, but was one of four men whose stomach was low in position; 62 was the only man who had achlorhydria on each of three occasions when he was examined, and he was one of seven men with unusually rapid emptying; 63 gave a low normal curve for his test-meal, but was one of the two men whose stomach was reported as low and hypotonic and slowly emptying. The other was not subjected to this series of tests, but did not look very robust. Of these men known or suspected to be specially unfit, three were found abnormal by their weight, three by their vital capacity, six by their pulse rate at rest, and two by their pulse rate after exercise.

There are seven other men (67, 69, 74, 76, 77, 78 and 79) who were picked out as abnormal in two tests. All of these were grouped as below the average; except for 79, who had been in bed for some days shortly before he was examined, and perhaps 69 they were not very fit and were taking no exercise of any sort.

Taking the tests one by one, nine men were judged abnormal by weight. Four of these were quite fit and were abnormal in no other way. Two were found abnormal in other tests and were very unfit. Three were found abnormal on other tests and were perhaps ill.

Fourteen men were judged unfit by their vital capacity. Six of these were normal by all other tests, three being average or above, and three being below average but not unfit. The other eight were judged abnormal by other tests as well, five being unfit and three perhaps ill. Ten men were at least 10 per cent. above the calculated value for their vital capacity. Nos. 1 and 67 were examined on two occasions and were 21 per cent. above their calculated value. No. 1 was extremely fit, but 67 was very unfit by every test and by everyone's opinion, though he certainly had good physique and would probably have been fit with training.

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Of three men 15-20 per cent. above, 16 was very fit, 18 was a long-distance runner but did not look particularly strong, and 40 was certainly not above the average. There were five men whose vital capacity was 10 per cent. above; two were athletes who were certainly fit (3·5); two were classed as above the average (24-35), but 24, as has been mentioned already, was probably not fit, and 50, though classified as average, had had two attacks of rheumatic fever and was unable to take any exercise. Of the ten men with high vital capacity, five were certainly very fit, but three were definitely unfit or abnormal in some other way.

Fifteen men were judged abnormal by their pulse rate at rest, of whom eight (46, 49, 50, 55, 56, 71, 72 and 80) were not picked out as abnormal by any other test but were certainly not very fit, although generally grouped as average. Two of the eight (50 and 80) only detected in this way were known to have been ill, and inspection of the response to other tests in Table X shows that all eight did badly, although not actually failing to reach the standard. The average weight was 8 per cent., and the vital capacity 5 per cent. below standard. In these men whose pulse at rest was 78 or above, there was more independent evidence of unfitness or abnormality than in those judged abnormal on the other tests.

Eleven men were classed as abnormal by the rate of their pulse after exercise. In all but two of these, other tests also showed the men to be abnormal and even then two were classed low as average and below average.

This confirms what was found by taking the groups as a whole. With weight and vital capacity only half the men, who were judged to be abnormal, showed any other evidence of abnormality. With the pulse rate at rest or after exercise a much larger proportion of the men judged to be abnormal were found to be so in other respects as well. By examining the pulse rate at rest and after exercise a good idea can be obtained if the man is unfit; by making use of all the tests together a very good idea can be obtained. It is disappointing to confess, but untruthful not to acknowledge, that reliance on any single test will lead to very serious errors. A better judgment would be given by a man of experience without tests than by a rigid adherence to any one, though the use of several is very helpful.

I wish to thank Professor Pembrey for his advice and kindly criticism, and Mr. R. R. Traill for his help in collecting many of the data.

## SUMMARY AND CONCLUSIONS

1. *Weight* can be estimated with fair accuracy from measurement of the chest and stem length and the use of Dreyer's tables, which are a great improvement on the old weight-height standards. In over 85 per cent. of these medical students the true weight does not differ by more than 10 per cent. from the calculated value. The weight is rarely more than 5 per cent. above standard, but variations of from — 5 to — 10 per cent. are of no significance and should not be included, as Dreyer suggests, as possibly abnormal. When it is more than 10 per cent. below, half the subjects are normal by other tests and by general opinion, and half show other signs of abnormality.

2. *Vital capacity* is more variable, but in about 65 per cent. of these men it lies within 10 per cent. of the calculated value. Where it is more than 10 per cent. above normal, there is other evidence of special fitness in about half the subjects, but in the other half the man is certainly not above the average, or definitely below it. Nearly a quarter of these students had a vital capacity 10 per cent. below the calculated value at their first attempt. Half of these were judged to be unfit or below the average on other grounds, but half were average or above.

3. In the majority of these students the observed weight and vital capacity are within 10 per cent. of the calculated values. Where they differ by more than this there is confirmatory evidence of some other abnormality in half of them. The examination is therefore of little value for individuals unless they are abnormal in more than one test, but in the examination of groups it is of value.

4. In addition to the usual variation of vital capacity, a special effort to reach a standard or beat some other reading adds at least a further 5 per cent., and this is of great importance when such a test is used in routine examinations, to decide if a man is fit for some particular occupation.

5. *The pulse rate at rest* is most useful to detect a man who is unfit, but it gives little guide to the degree of fitness, as it becomes slower as the weight increases. In this series of eighty students no one with a pulse rate of 78 or over is above the average fitness and only four can claim even average fitness. The other eleven are below the average, generally very much so, or definitely ill.

6. The pulse rate at rest is influenced by the weight of the subject, the average rate varying from 76 for the lighter to 60 for the heavier men. Apart from the effect of weight it

is also slower in the fit, varying from 65 in the athletes to 75 in those below the average.

7. The pulse rate in the first half-minute after the moderate exercise here considered depends mainly on the rate at rest, but the speed at which it returns to the resting figure is another valuable guide to a man's degree of fitness and varies with slight changes in his condition. Reliance on this test alone, as on the others described, would lead to some serious errors.

8. The pulse ratio as originally described is not satisfactory, because the unfit man with a high pulse rate at rest has an artificial advantage.

9. The pulse rate at rest and after exercise differs more in the various groups than do the weight, vital capacity or pulse ratio. As would be expected from this, abnormalities in these tests are more often an accurate guide to a man who is unfit than are the other tests.

10. Estimates of physical fitness by several observers agree more closely with each other than do the results of these various tests. A man who had experience in using tests for vital capacity or changes in the pulse after exercise would find them of value in helping him to estimate physical fitness. A man who was bound by the result of a single test would be at a great disadvantage compared with the man who was able to make general observations and conclusions.

But where a man is judged to be abnormal by two or more tests, it is almost certain that he is very unfit and possible that he is ill. To this extent the tests are valuable.

11. All the tests show a greater difference between the average men and the men who are unfit than between the average men and the athletes. Success in athletics and other pursuits requiring both physical and mental qualities depends on factors which are not readily discovered by any test except actual trial at the occupation in question.

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## APPENDIX

TABLE XI.

RELATIONSHIP OF WEIGHT, RATE OF RESPIRATION AND PULSE.

Animal.	Weight in grams.	Average rate of heart beat at rest.	Rate of respiration.
1. Goldfinch . . . . .	16	920	—
2. Canary . . . . .	20	1,000	100
3. Sparrow . . . . .	24	800	90
4. Mouse . . . . .	25	700	—
5. Greenfinch . . . . .	26	740	—
Average 1-5 . . . . .	22	830†	—
6. Young kingfisher . . . . .	42	440	—
7. Pigeon . . . . .	300	185*	30
8. Parrot . . . . .	430	320	—
9. Guinea pig . . . . .	400	300	—
Average 8-9 . . . . .	415	310†	—
10. Duck . . . . .	2,000	240	—
11. Rabbit . . . . .	2,000	205	55
Average 10-11 . . . . .	2,000	222†	—
12. Baby at birth . . . . .	3,300	136	44
13. Cat . . . . .	4,000	160†	24
14. Hare . . . . .	2,500	64*	—
15. Dog . . . . .	8,000	120†	—
16. Boy at 10 years . . . . .	25,000	90	24
17. Large dog . . . . .	30,000	85†	15
18. Sheep . . . . .	40,000	75†	—
19. Pig . . . . .	60,000	75†	—
20. Adult man . . . . .	70,000	70†	16
21. Bullock . . . . .	500,000 (?)	48	—
22. Horse . . . . .	600,000 (?)	37	11

† The curve in Fig. 1 is drawn from these points which includes all animals except man and the two marked \*.



TABLE X—continued.

No.	Age.	Stom. length.	Chest.	Observed weight.	Calculated weight.	Percentage difference.	Observed vital capacity.	Calculated vital capacity.	Percentage difference.	Re-ting pulse.	Pulse after exercise in half-minutes.	"Pulse ratio."	Regular exercise taken.
18	26	92	88	58.5	67.8	-14	5100	4360	+17	72	60	2.7	Running.
19	20	92	91	64.1	71.0	-8	4000	4510	-11	68	64	3.0	Rugby.
20	30	90	94	66.2	72.2	-8	4600	4550	+1	74	68	3.2	Soccer.
21	31	89	92	68.6	68.2	0	4500	4370	+3	60	54	2.8	Hockey.
22	25	92	90	66.5	70.0	-5	4500	4460	+1	60	58	2.6	Soccer.
23	28	86	83	57.1	56.2	+2	3800	3800	0	56	52	2.8	Hockey; soccer.
24	19	92	86	58.5	66.8	-13	4900	4320	+14	80	67	2.6	Boxing; soccer.
25	19	89	93	68.9	69.7	-1	4200	4460	-6	74	70	3.3	Tennis; golf.
26	27	86	97	68.0	71.8	-4	4100	4500	-9	72	66	3.0	Rowing.
27	35	91	94	67.7	73.9	-8	4600	4640	-1	69	54	2.8	Soccer.
28	20	86	83	56.5	56.3	0	3600	3820	-6	68	54	2.4	Bicycling.
29	33	91	93	66.7	72.1	-7	4200	4550	-8	76	60	2.6	Rugby.
30	18	88	86	58.0	61.2	-5	4400	4040	+9	66	63	3.2	Nothing.
31	20	95	91	67.5	74.7	-9	4700	4670	0	70	62	2.8	Soccer; boxing.
32	21	87	80	54.2	54.5	0	3400	3730	-9	76	56	2.8	Running.
33	19	95	86	64.0	69.4	-8	4300	4440	-3	72	56	2.5	Rugby.
34	17	93	80	59.6	61.6	-3	3900	4070	-4	64	50	2.4	Rugby.
35	20	98	98	74.0	87.0	-15	5800	5220	+11	66	54	2.7	Various.
36	23	94	89	67.4	71.3	-6	4100	4520	-9	68	54	2.6	Tennis.
Av.	24	90.9	89.1	63.8	67.7	-6	4310	4360	-1	69.0	60.0	2.72	



TABLE X—continued.

No.	Age.	Stom. length.	Chest.	Observed weight.	Calculated weight.	Percentage difference.	Observed vital capacity.	Calculated vital capacity.	Percentage difference.	Resting pulse.	Pulse after exercise in half-minutes.	"Pulse ratio."	Exercise.	2nd minute.
37	18	90	83	61.6	60.5	+1	4000	4920	0	68	60	3.0	Nothing.	101
38	24	93	96	73.4	78.7	-7	5200	4860	+7	69	56	2.7	Nothing.	90
39	20	84	83	53.0	54.8	-3	3800	3740	-1	76	70	2.8	Nothing.	81
40	21	90	85	62.0	62.5	0	4900	4110	+19	60	60	3.1	Nothing.	81
41	18	89	90	62.0	66.5	-7	3900	4300	-10	72	64	2.7	Tennis.	92
42	20	92	86	64.3	65.7	-2	4500	4260	+6	76	51	2.4	Tennis.	75
43	19	88	82	61.8	57.3	+8	3300	3860	-14	58	55	3.1	Nothing.	76
44	23	93	91	67.0	72.2	-7	4500	4560	-1	59	60	3.2	Nothing.	82
45	22	92	87	65.4	66.8	-2	3800	4300	-11	68	68	3.3	Nothing.	102
46	23	87	86	59.6	60.2	-1	3700	4000	-7	79	59	3.3	Nothing.	99
47	19	85	87	61.8	59.2	+4	3600	3950	-9	68	70	2.7	Nothing.	87
48	18	91	85	59.0	63.6	-7	4500	4160	+8	72	51	2.6	Nothing.	89
49	20	95	87	63.7	70.4	-9	4500	4480	0	80	67	2.7	Nothing.	103
50	20	87	84	56.9	58.7	-3	4500	3930	+14	88	59	2.4	Nothing.	67
51	26	95	90	71.7	73.6	-3	5100	4630	+10	55	54	2.9	Nothing.	96
52	29	87	87	61.2	61.2	0	4300	4050	+6	68	61	3.1	Nothing.	82
53	19	90	92	70.5	70.0	0	4900	4460	+10	70	55	2.6	Nothing.	84
54	19	91	90	61.0	68.8	-11	4500	4410	+2	60	56	3.1	Nothing.	110
55	20	87	96	66.5	71.3	-7	4200	4520	-4	81	76	3.0	Nothing.	107
56	20	95	88	65.6	71.5	-8	4700	4530	+4	82	72	3.6	Tennis.	118
57	19	85	80	55.7	53.0	+5	3400	3650	-7	68	72	3.7	Nothing.	118
58	19	94	91	64.1	73.5	-13	4100	4620	-11	71	73	2.8	Nothing.	66
59	21	96	92	68.6	77.2	-11	5000	4780	+4	57	54			
Av.	21	90.2	87.7	63.3	65.6	-3.5	4300	4260	+1.0	70.3	62.0	2.93		91

TABLE X—continued.

No.	Age.	Stem length.	Chest.	Observed weight.	Calculated weight.	Percentage difference.	Observed vital capacity.	Calculated vital capacity.	Percentage difference.	Resting pulse.	Pulse after exercise in half-minutes.	"Pulse" ratio.	Exercise.	2nd minute.
60	21	90	88	60.2	65.5	- 8	4700	4250	+ 10	65	48	2.6	Nothing.	76
61	23	89	90	65.6	66.5	- 2	3800	4300	- 11	68	60	3.1	Nothing.	101
62	22	95	85	59.5	68.4	- 13	4400	4390	0	90	84	3.3	Nothing.	136
63	19	97	90	73.7	77.8	- 5	4100	4820	- 15	62	72	3.8	Soccer.	106
64	21	85	88	55.5	60.2	- 8	4300	4000	+ 7	76	70	3.5	Nothing.	118
65	22	91	86	62.6	64.6	- 3	4200	4210	0	70	54	2.5	Nothing.	79
66	32	94	109	92.5	97.0	- 5	4800	5640	- 14	81	64	2.7	Nothing.	99
67	23	95	101	80.9	86.9	- 7	6300	5220	+ 21	78	82	3.6	Nothing.	125
68	21	84	81	53.8	52.3	+ 3	3100	3610	- 14	68	56	2.7	Nothing.	79
69	20	89	92	64.3	68.8	- 6	3800	4410	- 13	75	77	3.4	Tennis.	122
70	26	92	92	72.0	72.2	0	4400	4560	0	66	64	3.0	Nothing.	87
71	20	89	87	57.0	63.3	- 10	4200	4150	- 2	82	76	2.7	Nothing.	110
72	25	91	90	66.5	68.8	- 3	4300	4400	- 11	82	63	3.3	Nothing.	104
73	30	88	99	75.4	76.0	0	4200	4730	- 14	71	66	2.5	Nothing.	107
74	23	86	93	82.1	66.8	+ 22	3700	4310	- 14	58	65	3.7	Nothing.	94
75	19	95	88	64.1	72.1	- 11	4000	4350	- 7	84	60	3.8	Nothing.	95
76	20	92	86	58.9	66.8	- 12	4000	4310	- 17	68	73	3.4	Nothing.	120
77	20	92	84	60.8	63.8	- 5	3400	4170	- 13	73	66	2.7	Nothing.	118
78	20	83	81	46.9	51.4	- 9	3100	3570	+ 10	86	65	3.5	Nothing.	106
79	22	88	87	61.9	62.3	0	4500	4110	0	78	83	2.4	Nothing.	125
80	21	92	85	64.0	64.7	- 1	4200	4210	0	80	58	3.1	Nothing.	78
Av. 22.5	90.3		90.3	65.6	68.2	- 4	4170	4380	- 5	74.5	65.4	3.10		101.5
No. 67, 66 omitted	90.0		88.3	63.4	65.7	- 4	4020	4270	- 6		56.0			

## AN INVESTIGATION INTO THE PHYSIOLOGICAL BASIS OF THE U-TUBE MANOMETER TEST

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THE U-tube manometer test, first introduced by Stephen Hales and lately elaborated by Wing-Commander. Martin Flack<sup>1</sup> for testing the efficiency of members of, and entrants to the Royal Air Force, has recently come into more general use, and is now applied during investigations into industrial efficiency. It was felt that there was a need of investigation into the physiological basis of the test, and for this reason the present research was commenced.

The apparatus comprises a mercury manometer, connected by pressure tubing with a mouthpiece; this latter is composed of thick glass and resembles a small filter funnel flattened in one plane.

The test is conducted as follows: The subject makes a deep expiration followed by the deepest possible inspiration, and then quickly introduces the mouthpiece between the lips and the teeth, which clasp the mouthpiece tightly, and blows through it until he is blowing against a pressure of 40 mm. of mercury. The mercury is raised to the required level as quickly as possible, for it is found to be much more strenuous to raise the mercury slowly. While the mercury is sustained at the level of 40 mm., the subject is warned not to allow ballooning of his cheeks, and of course does not introduce his tongue into the mouthpiece. A few subjects tend to lose air through the nose, and these are fitted with a nose-clip, but many subjects prefer not to use the clip, because the discomfort caused by it lowers the time during which the mercury can be sustained.

The mouthpiece described above is of rather a large size, and causes early tiring of the orbicularis oris muscle. This was prevented in later experiments by replacing it by a small cigarette-holder, and it was found that this could be more readily gripped with less discomfort.

The test may be applied in one of two ways, either (a) the

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maximum time during which the subject can sustain the mercury is tested by a stop-watch, or (b) the mercury is held at the level for a definite period (*e.g.* 20 seconds) and the pulse rate during and after the test is compared with that before the test.

#### METHOD OF INVESTIGATION

Digital examination of the radial pulse during and after the test shows that the rate varies considerably from second to second, and so the method of previous observers,<sup>1, 3</sup> who counted the pulse rate during consecutive five-second periods, was discarded in favour of a much more accurate graphic record. This can be obtained as follows. A blood-pressure armlet is connected with a tambour, the membrane of which is made of the inner tubing of a bicycle tyre. When the pressure inside the armlet is raised to about 60 mm. of mercury, the recording lever of the tambour gives a pulse record on the smoked paper of a revolving drum, and underneath is a time record. The commencement of the test is indicated by an automatic electric signal, made by the mercury as it is raised in the U-tube. The records are analysed accurately, and the pulse rate calculated from the duration of each beat plotted on squared paper.

In the later experiments systolic blood-pressure records were taken simultaneously by another observer. For this purpose a Riva Rocci blood-pressure apparatus was fitted to the brachial artery of the other arm of the subject, with the addition of a compressible rubber bulb in the side circuit. By varying the pressure on this, the variations in the systolic blood-pressure were estimated by radial digital examination, and were called out by the observer, who signalled the moment at which the reading was taken by an electric signal controlled by his foot. The figures were afterwards entered on the graph. With a little practice the accuracy of the blood-pressure records was probably always within 5 mm. of the correct reading.

#### RESULTS

The subjects investigated included men of very varied physical fitness, and the age ranged from 15 to 35 years. A typical result is shown in Fig. 1.

The variations seen are :—

*Before the test.*—(1) Three to five seconds before the test commences, the pulse rate tends to fall below the normal, followed by—

(2) A considerable rise in pulse rate occurring immediately before the mercury is raised.

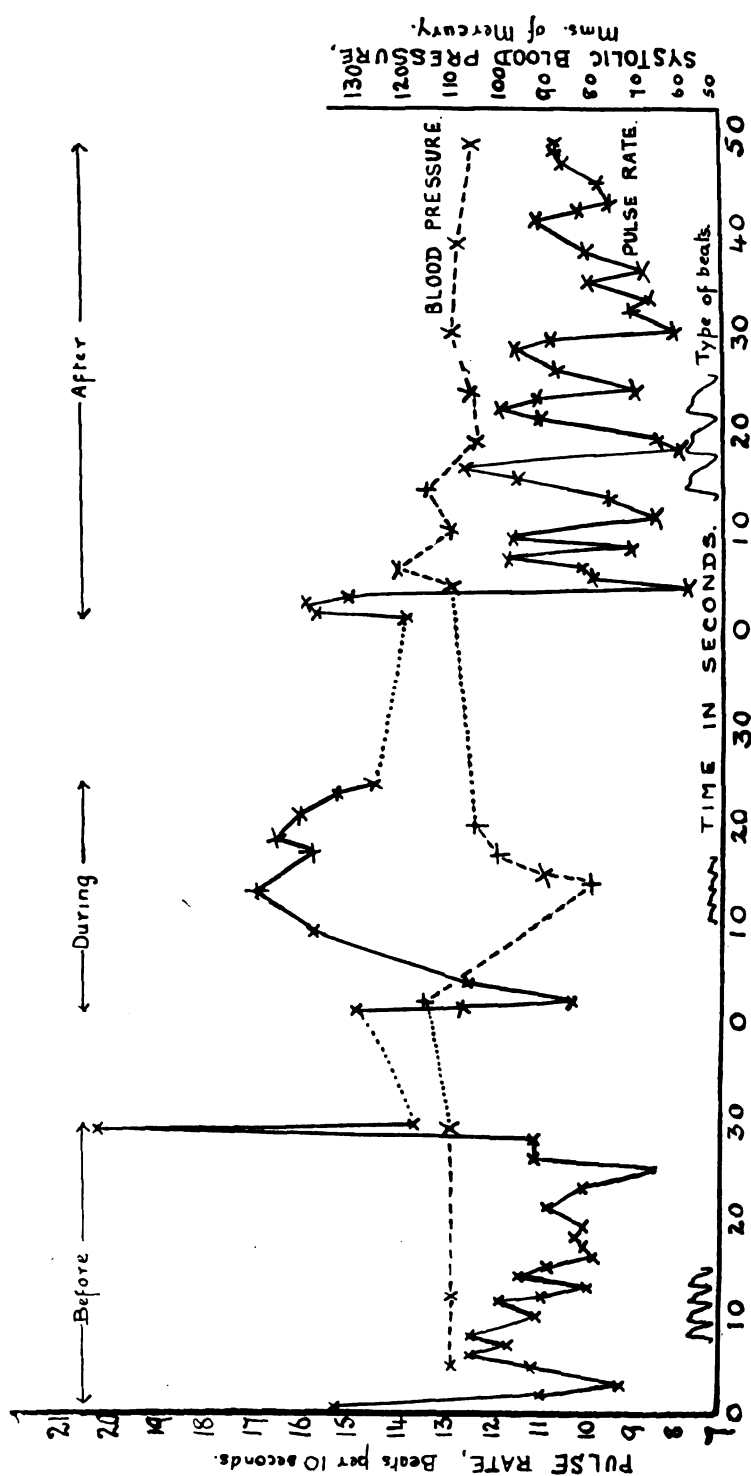


FIG. 1.  
28.2.22. Graph 39, ii. Subject, C.A.N.H.

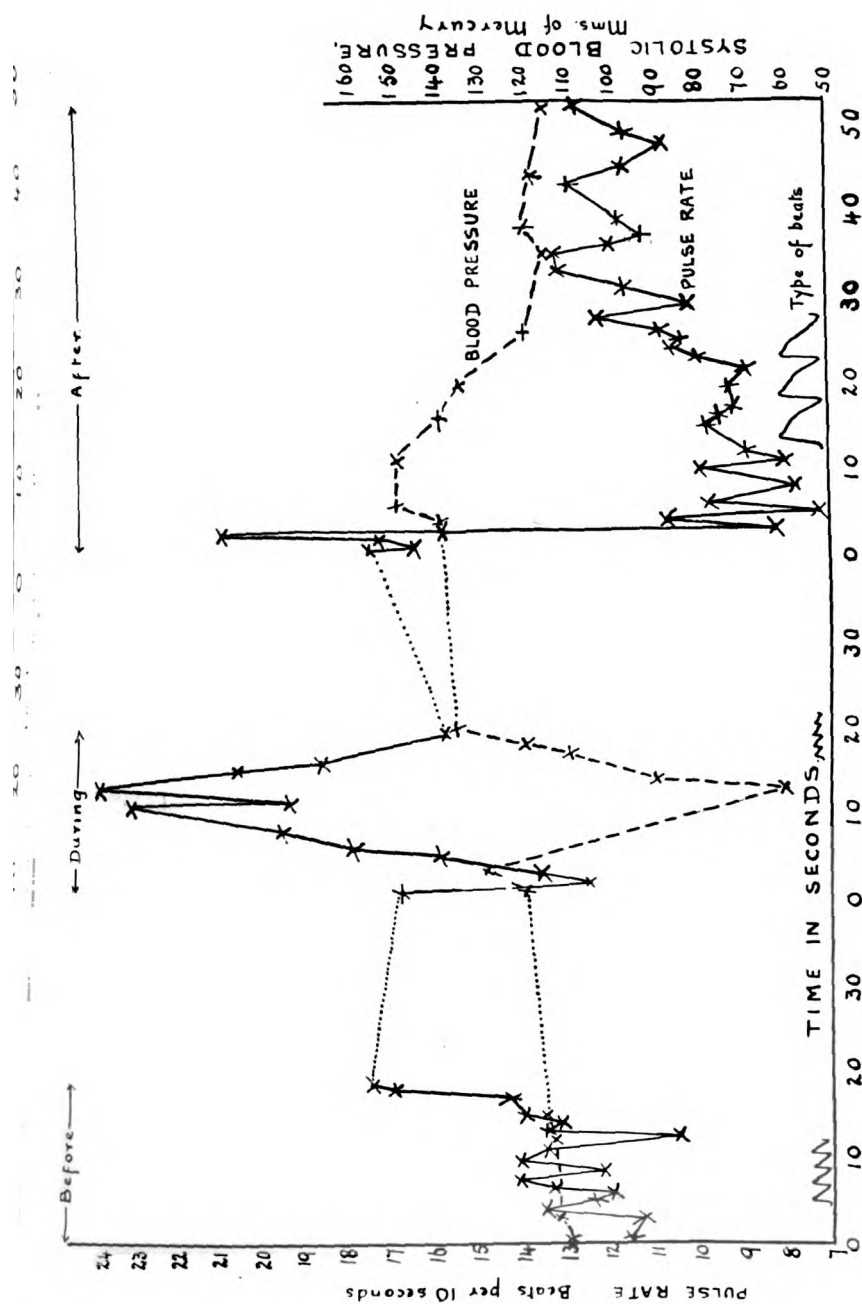


FIG. 2.

13.3.22. Graph 60, ii. Subject, C. M.

*During the test.*—(1) The pulse rate rapidly returns towards the normal during the first two or three seconds of the test, this being associated with a transitory rise in blood pressure of about 5 mm. of mercury.

(2) Then the pulse rate steadily rises for the next five to twelve seconds coincident with a considerable fall of blood-pressure (to about twenty to thirty seconds below normal). During this period the beats of the pulse as recorded on the graph become very feeble in comparison with those at rest.

(3) During the remainder of the test the pulse rate tends to recover and the systolic blood pressure to rise, that is to say, there is a recovery towards the normal values—at the same time the beats become more powerful.

*After the test.*—(1) The immediate effect is a short sharp rise in pulse rate for the first two to four seconds, followed by—

(2) A dramatic fall in pulse rate to a value much below the normal. Corresponding to this is a small but sustained rise in blood pressure at about 10 mm. above the resting value. The beats are now seen to be very slow but powerful to an extreme degree. There is also considerable sinus arrhythmia.

(3) The pulse rate gradually rises and the blood pressure falls to the resting value.

The variations from this type of result are merely a matter of degree. Thus in subject C. M., who is less fit physically than is C. A. N. H., the variations both in pulse rate and in blood pressure are much more extreme (Fig. 2). While in F. C. S., who is physically very well developed, and is a good athlete, but small variations are noticed. And similarly variations in the characters of the beats are to be noticed.

It is necessary to emphasise that there is a considerable “practice effect” followed by a “fatigue effect” when a series of tests is performed with intervals of about three minutes.

#### ANALYSIS OF RESULTS

*Before the test.*—The factor influencing the variations in pulse rate and blood pressure may be of respiratory or of psychological origin. It was noticed that the deep inspiration for the test began three to four seconds before the marking of the signal indicating the commencement of the test. That the initial fall in pulse rate is due to the previous phase of expiration is shown by Fig. 4, and this agrees with well-known observations on animals. That the excitement of having to perform a task is accompanied by a rise of pulse rate was first shown by Krogh, but that this is not the chief factor in the

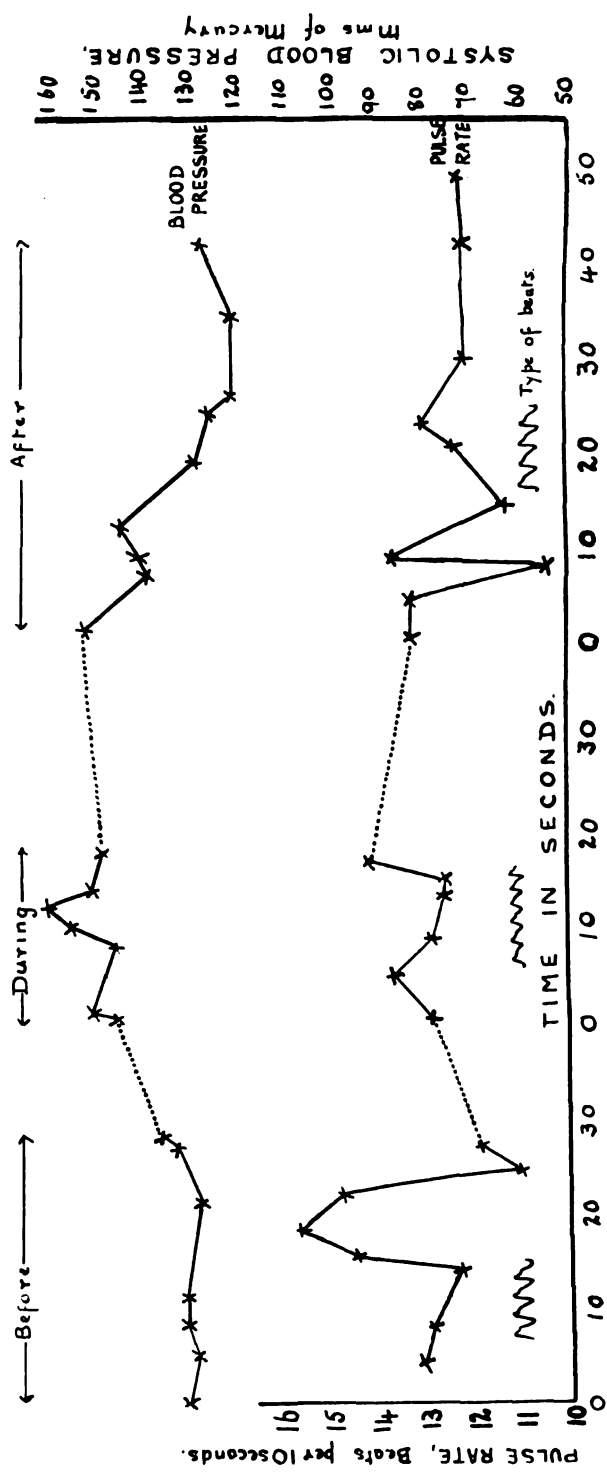


FIG. 3.  
26.1.22. Graph 29, ii. Subject, F. C. S.



rise in pulse rate immediately before the test is shown in two ways. If the test is performed at the end of an expiration without a previous deep breath, then the rise in pulse rate is present to the extent of only two beats per ten seconds instead of eight beats per ten seconds. Secondly, a rise similar to that observed when the test was performed was found as a result of deep breathing when no test followed. The rise in pulse rate, therefore, is largely due to the sudden deep inspiration,

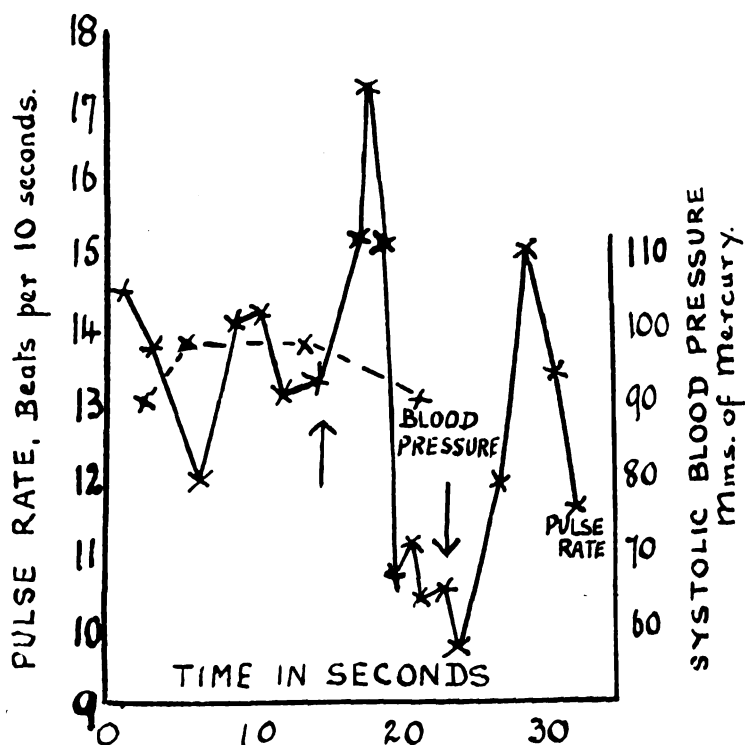


FIG. 4.

28.2.22. Graph 40, iv. Subject, C. A. N. H. The first arrow indicates the commencement of a slow deep inspiration; and the second arrow of a slow deep expiration.

and, moreover, was found to be proportional to the depth of the inspiration.

*During the test*, we realise that there are two groups of factors to consider: (a) those due to the gaseous interchanges occurring in the lungs, and (b) those due to the mechanical factors operating in the thorax and in the abdominal and peripheral vessels.

During the cessation of respiration which occurs with the period of the test, the tension of carbon dioxide in the lungs

is increasing while the tension of oxygen is diminishing. Both of these are capable of producing variations in vagal tone, as is shown, for instance, in asphyxia. In the case of the U-tube manometer test, however, neither excess of carbon dioxide, nor lack of oxygen has any marked influence on either the pulse rate or the systolic blood pressure. These showed the same variations, whether the subject was breathing expired air or atmospheric air. (The nature of the gas breathed was, of course, unknown to the subject.) Secondly, when the subject breathed 96 per cent. oxygen, the result was a smaller average and absolute rise in the pulse rate during the test, but with a greater degree of subsequent slowing. The general shape of the curves is normal (Table I).<sup>1</sup> So it may be concluded that gaseous interchanges in the lungs, during and after the test, do not play any appreciable part in causing the changes of pulse rate observed.

Turning then to the mechanical factors operating in the thorax and abdomen, we find that the initial fall in pulse rate and rise in systolic blood pressure are largely due to the recovery from the high pulse rate due to the deep inspiration. Thus, similar variations, but of less extent, occur when the breath is

TABLE I.

Breathing.	Average before test.	Average during test.	Highest value above normal.	Recovery.	Average after test.	Lowest value below normal.	Average of experiments.
<i>Subject, W. D. H.</i>							
Graph 4. Oxygen.	12.9	13.4	15.2	14.4	10.3	9.1	4
" 3. Air.	11.4	13.7	15.9	15.1	11.4	9.5	3
" 8. Oxygen.	11.7	12.9	14.7	13.9	11.0	8.9	2
" 8. Air.	11.6	12.4	13.9	13.5	11.9	10.3	2
<i>Subject, A. C. W.</i>							
Graph 6. Oxygen.	15.1	15.3	18.3	13.4	11.4	10.3	2
" 6. Air.	13.9	14.9	18.3	13.0	12.0	11.0	2

held after a deep inspiration has been taken, but no test follows. A contributing factor is the forcing of blood from the lungs to the left heart as the capacity of the lungs is decreased by raising the mercury. This is shown by the smaller fall of pulse rate and rise of blood pressure which occur when the test is performed without a preceding deep inspiration.

The more important variations occurring during the test are the rise and recovery of the pulse rate and the fall and recovery of the systolic blood pressure. Of these the primary factor is probably the blood pressure, for the changes in this seem to precede the changes in the pulse rate. The explanation of these effects is clear if we consider the changes occurring in

the thorax and abdomen during this period. We will classify these mechanical changes under two headings.

- Firstly, with the necessary cessation of respiration during the test, the intermittent action of the cardio-pulmonary pump ceases, and as a result of raising the mercury, the normal negative intra-thoracic pressure becomes a positive pressure. Since it is largely by virtue of this negative pressure that venous return to the thorax is brought about, raising the mercury imposes a resistance to venous return to the thorax. This is appreciated in the head as the unpleasant "bursting sensation."
- But if the intra-abdominal pressure during the test is also raised, and if it reaches a higher level than the intrathoracic pressure, then blood will still be forced towards the heart from the abdomen.

The relative pressure in the thorax and abdomen have been studied by placing non-elastic rubber balloons in the œsophagus and stomach respectively. The results obtained are shown in Table II, and show that throughout the test the pressures remain steady and always with a positive pressure in favour of the abdomen. So that during the period of holding up the mercury blood will be able to reach the heart from the abdomen, but will not do so from the limbs and head and neck.

Secondly, there is the collapsibility of the vessels to consider. In contrast to the pressure required to collapse the arteries, we know that the thin-walled veins will collapse completely when the pressure outside them is only slightly greater than that inside, and it seems probable that the capillaries would do so also.

TABLE II.

(Experiments carried out on W. W. P., who, as a result of long practice, can tolerate well the balloon in the œsophagus or stomach. His pulse response to the U-tube test was shown to conform to the above description of the normal response.) The readings were taken with a water manometer, but these results have been converted into the equivalent pressures in mm. of mercury, and allowance has been made for the pressures already existing in the bags, so the values are absolute values.

*(Esophagus.)*—The balloon was 2-3 ins. above the cardia. A sudden deep inspiration causes a negative pressure of 15 mm.

Holding up the mercury U-tube to a height of 40 mm. for twenty seconds.

	After a deep inspiration.		After normal expiration. (No deep breath taken for test.)	
	I.	II.	I.	II.
Deep breath . . . . .	- 10 mm.	- 8 mm.	—	—
Holding up mercury :				
(a) At commencement . . .	+ 28.5 ..	+ 24 ..	+ 25.5 mm.	+ 20 mm.
(b) Later steady at . . .	+ 25.5 ..	+ 18 ..	+ 25.5 ..	+ 18 ..

The readings obtained when the subject was making ordinary quiet respirations showed a negative pressure of from  $-1$  to  $-4$  mm.

*Sucking* the mercury of the U-tube to a height of 40 mm. caused a sudden negative pressure of 29 mm. of mercury, which soon became steady at 32.5, 33.5 and 35.5 mm. in consecutive experiments.

*Stomach*.—Pressure in bag was  $+5$  mm.

Deep thoracic breathing gave no alteration in stomach pressure, whereas deep abdominal breathing gave a positive pressure of 22 mm., but this depended on the rapidity of inspiration. When sustained it remained at a positive pressure of 10 mm.

*U-Tube manometer test held up to a height of 40 mm. for twenty seconds.*

	I.	II.	III.	IV.
1. Deep inspiration . .	$+22$ mm.	$+25$ mm.	$+22$ mm.	$+28$ mm.
2. Blow up mercury . .	$+30\frac{1}{2}$ „	$+36\frac{1}{2}$ „	$+38$ „	$+40$ „
3. 5 seconds later . .	$+39\frac{1}{2}$ „	—	$+45\frac{1}{2}$ „	$+34$ „
4. 5-20 seconds steady at .	$+30\frac{1}{2}$ „	$+30\frac{1}{2}$ „	$+36\frac{1}{2}$ „	$+34$ „

*U-Tube manometer test held up to 40 mm.*

	After a deep inspiration followed by a normal expiration.		After deep expiration, no previous inspiration as in I. and II.
	I.	II.	III.
Blow up mercury . .	$+51\frac{1}{2}$ mm.	$+57\frac{1}{2}$ mm.	$+48\frac{1}{2}$ mm.
Immediate short drop to .	$+39\frac{1}{2}$ „	$+53$ „	$+39$ „
Gradually rising, or steady throughout at . .	$+57\frac{1}{2}$ „	$+45\frac{1}{2}$ „	$+45$ „

Blood will collect on the arterial side of the compressed vessels, until the increase of pressure on the arterial side is sufficient to reopen first the capillaries and then the veins.

We can now see the reason \* for the fall of systolic blood pressure and the rise in pulse rate at the beginning of the period of holding up the mercury. The blood flow to the right side of the heart has diminished, and so the systolic blood pressures falls and the pulse rate quickens (Marey's Law) in an effort to keep up the blood pressure. (This is an explanation

\* The systolic blood pressure in the pulmonary artery is given as 15-20 mm. of mercury,<sup>4</sup> and that in the lung capillaries must be less than this value. It appears, therefore, that the capillaries of the lungs will be partially collapsed when the mercury is raised and it is not until the veins opening into the right heart are dilated again by the accumulated blood that the capillaries of the lungs can also be opened and so blood reach the left heart.

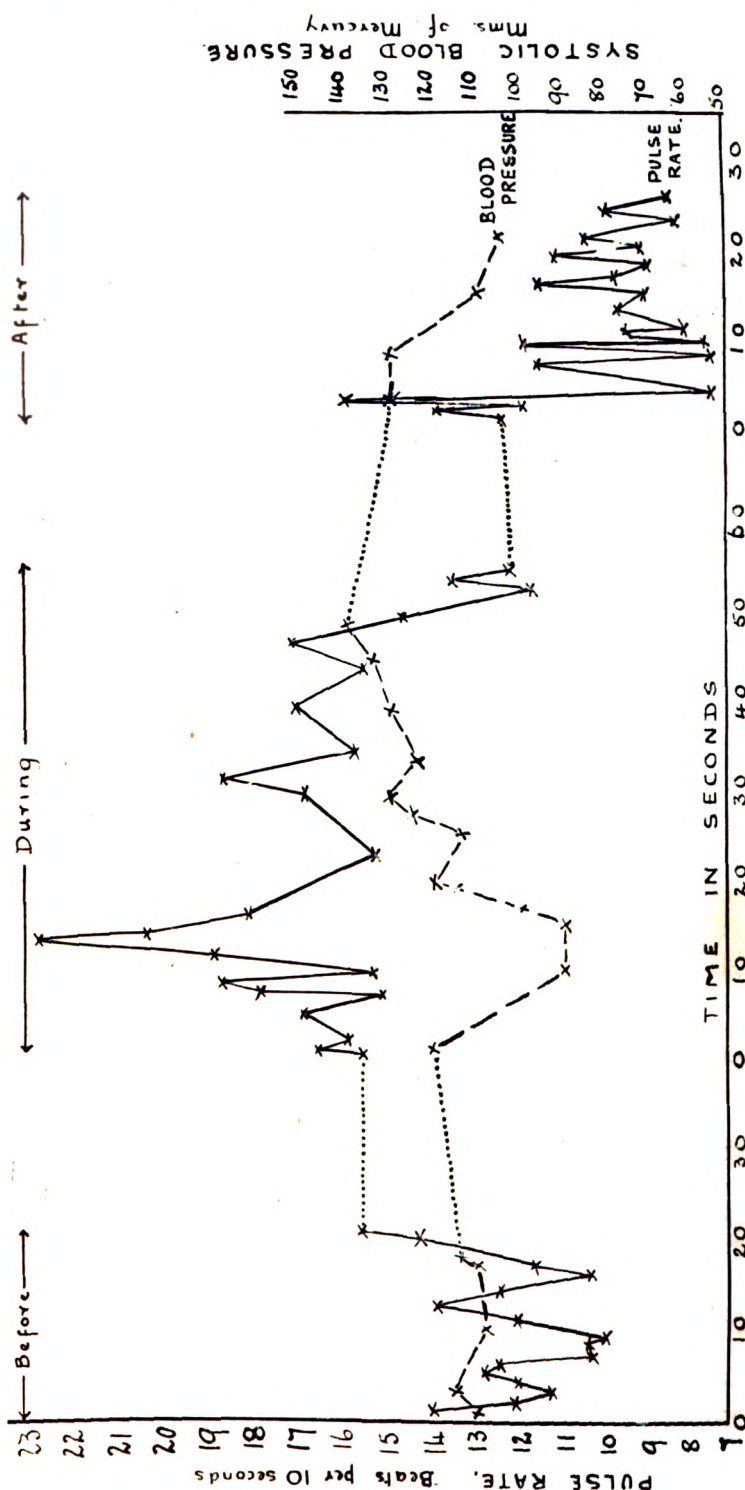


FIG. 5.  
1.3.22. Graph 43, iii. Subject, C. A. N. H. In this experiment the mercury was sustained for fifty-five seconds instead of the usual twenty seconds.

of the feeble rapid pulse at this phase of the test.) This continues until the blood has accumulated sufficiently to reopen the veins, when, the venous flow to the heart increasing, the systolic blood pressure will rise and the pulse rate can return towards normal (Marey's Law). This rise in systolic blood pressure and fall in pulse rate is found to continue when the mercury is held up for more than twenty seconds, so that finally the systolic blood pressure may be 20-30 mm. above normal at the 40th, or 50th second, and the pulse rate at the resting value.

That the fall in systolic blood pressure is due to a diminution of venous return to the heart is confirmed by x-ray evidence (as far as this can be accepted). The whole heart assumes a very small bulk during the test, suggesting that its cavities are considerably depleted of blood.

Since the abdominal and thoracic veins are compressed by the external pressure to which they are subjected, then the better the tone of the splanchnic vessels and of the abdominal walls, the smaller is their capacity and so the more quickly will the systemic veins become patent. The importance of good abdominal tone is well established by the following experiments. In the first place the test was performed by the same subject, who alternately wore and discarded a very tight abdominal bandage.

TABLE III.  
PULSE RATES (beats per ten seconds).

	Average before.	Average during.	Highest value above normal.	Recovery.	Average after.	Average lowest value.	Average of experiments.
<i>Subject, W. D. H.</i>							
Abdominal belt worn.							
Graph 23 . . .	10.7	11.8	13.3	12.4	10.8	9.5	3
Normal.							
Graph 23 . . .	10.9	13.7	15.5	15.5	10.7	9.5	2
<i>Subject, C. A. N. H.</i>							
Abdominal belt worn.							
Graph 37 . . .	14.7	18.3	21.0	18.0	12.8	7.6	3
Normal.							
Graphs 36 and 38	14.6	18.6	20.7	18.4	10.9	8.4	5

In the case of subject W. D. H. there is seen a very definite effect, the variations in the pulse rate being considerably less when the belt was worn. Unfortunately blood-pressure observations were not made. With C. A. N. H. the improve-

ment is much less, as shown by the pulse rate and the variations in blood pressures being the same in the two cases.

In the next experiment the tight abdominal bandage was released about midway during the period of holding up the mercury. The pulse rate, which was tending to rise less rapidly, suddenly rose to a much higher value, coincident with a rapid fall in blood pressure.

Further, we were fortunate enough to have in the wards of Guy's Hospital a boy (æt. 17) who had a congenital absence of his anterior abdominal wall musculature. By kind permission of Dr. Hurst, he was investigated from a physiological standpoint.<sup>5</sup> Although he was extraordinarily well compensated he could not hold up the mercury for longer than twenty-one seconds when his Curtis's abdominal support was not worn, but with the belt he maintained the mercury for thirty-one seconds. Graphic records showed that when he held the mercury up for twenty seconds, his pulse rate during the test rose ten beats per minute higher when his belt was not worn, and the pulse had less tendency to recover.

TABLE IV.

PULSE RATE (beats per ten seconds).

	Average before.	Average during.	Greatest rise above normal.	Recovery.	Average after.	Greatest below normal.	Average of experiments.
<i>Subject, E. J.</i>							
With Curtis's belt	16.4	18.3	21.6	18.6	15.2	11.1	4
Without belt	14.0	17.5	19.5	18.1	13.3	8.9	2

A further point in confirmation of the importance of the action of the abdominal wall in raising the intra-abdominal venous pressure is shown when we study the movements of the abdominal wall during the test.

We find that although the diaphragm seen by x-ray remains absolutely stationary throughout the holding period, the abdominal wall is continually contracting on its contents. So does the thoracic, compensating for the extra amount of gas which is being dissolved into the blood.\*

✓ *After the Test.*—The first variation is a sudden sharp rise in the pulse rate, coinciding with a smaller rise in systolic blood pressure. This is doubtless due to the sudden removal of the increased intra-thoracic and intra-abdominal pressure. The

\* We have not investigated to what extent venous inflow occurs from the head and neck and limbs to the heart during the period of sustaining the mercury.

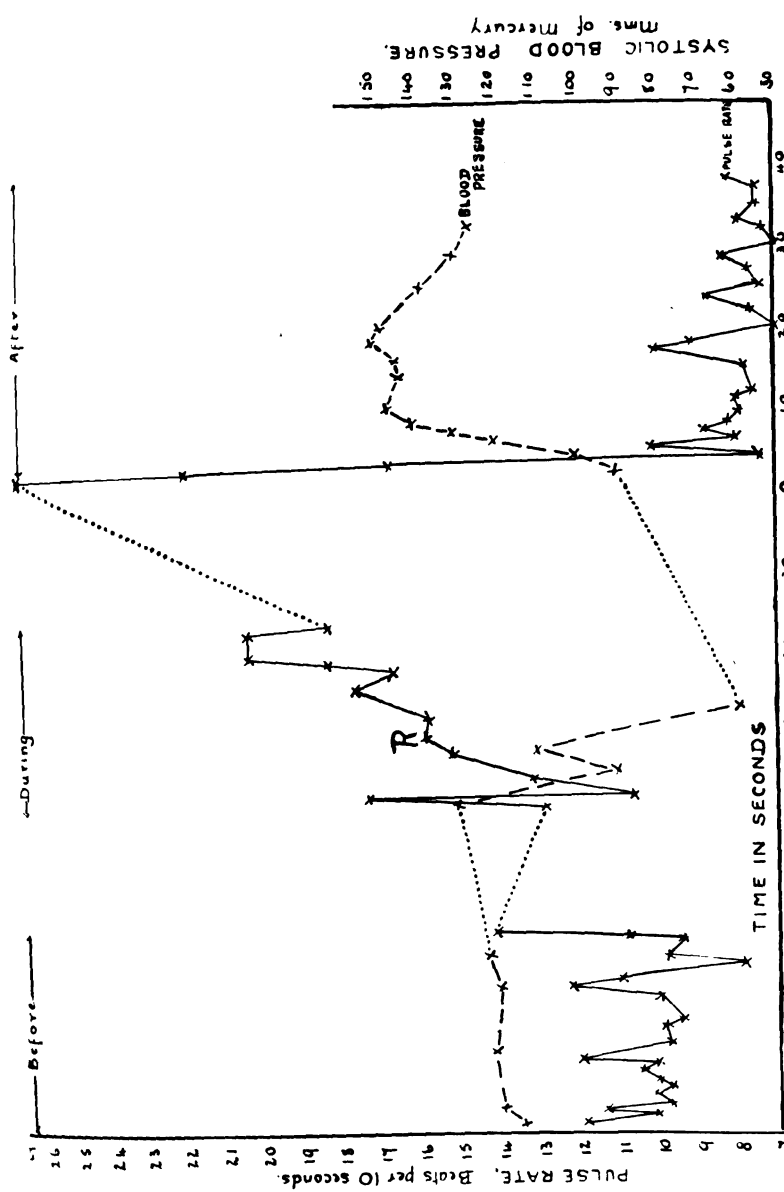


FIG. 6.

1.3.22. Graph 45, ii. Subject, C. A. N. H. The subject was wearing a very tight abdominal binder which was suddenly released during the period of sustaining the mercury at the point marked R.



blood which has accumulated in the thorax and abdomen, together with that from the limbs and head, is suddenly flooded to the right side of the heart, producing a rise in pulse rate by means of Bainbridge's reflex. This blood quickly reaches the left ventricle, the output of which is considerably increased, producing a rise in blood pressure. This rise in blood pressure is the cause of the variations which follow. We have already seen that these consist of a dramatic fall in pulse rate to a value below normal, while the systolic blood pressure is maintained. The strong slow beats with marked sinus arrhythmia which follow are an expression of the reflex vagal slowing of the heart, in order to prevent an excessive rise of blood pressure (Marey's

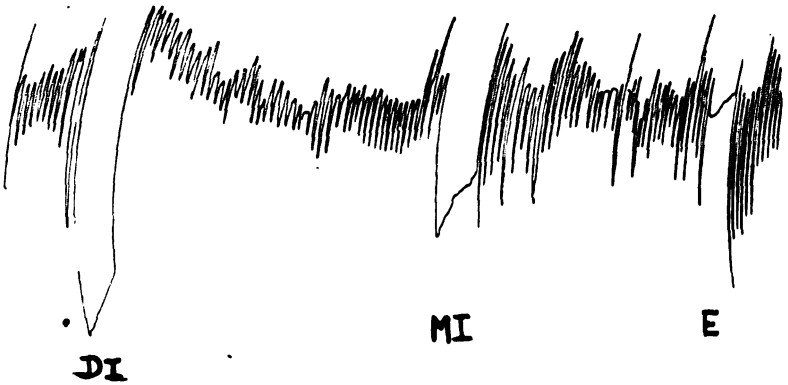


FIG. 7.

7.2.22. Subject, W. D. H. Record of the movements of the abdominal wall at the level of the umbilicus during the test when performed for twenty seconds after (a) a deep inspiration (D. I.), (b) a medium inspiration (M. I.), and (c) at the end of a normal expiration (E.). Inspiration is indicated by a downward movement of the level. Similar graphs were obtained at the level of the apex beat and of the axilla.

Law). It follows from this that those subjects who have poor tone in their abdominal wall and splanchnic vessels have a large capacity in these vessels, and thus have accumulated more blood in these areas to overcome the collapsing force exerted on the vessels during the test. These subjects have a greater fall in systolic blood pressure and rise in pulse rate at this period and a greater amount of blood dammed back in their abdominal vessels at the end of the test. They have a greater flooding of the heart when the mercury is released, with consequently a greater rise in systolic blood pressure and fall in pulse rate after the test, but the rapidity of recovery

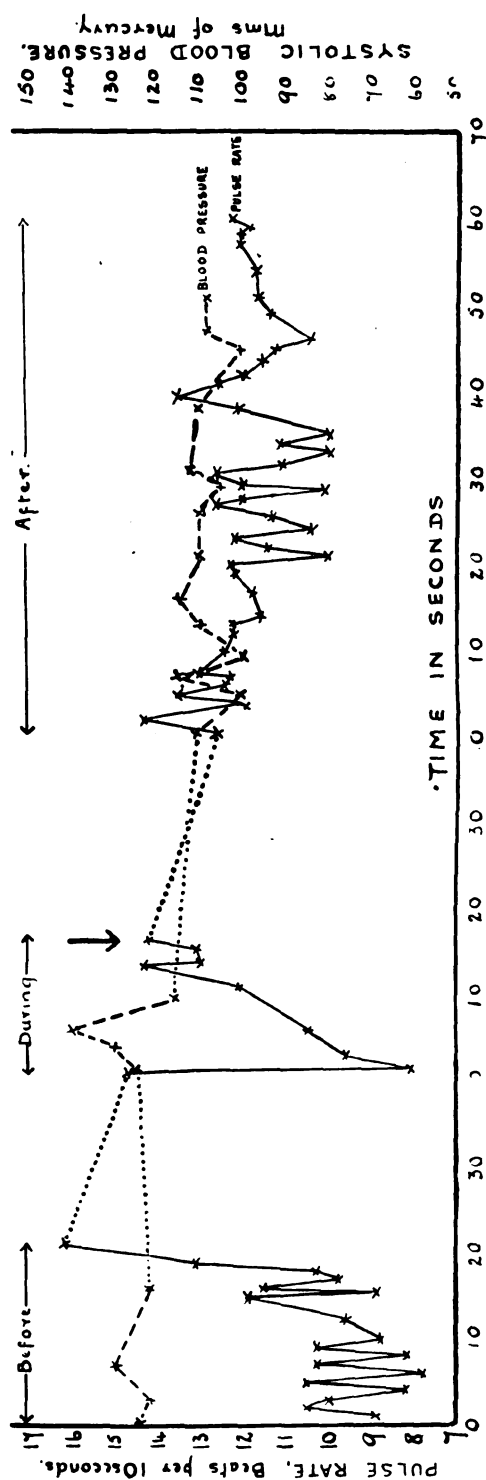


FIG. 8.

13.22. Graph 44, i. Subject, C.A.N.H. The subject was wearing a very tight abdominal binder which was suddenly released at the end of the period of sustaining the mercury, as indicated by the arrow.

towards normal will be slower, producing a still greater *average* fall in the pulse rate below the normal. Yet again, we find that when the same subject performs the test several times, on the occasions that the pulse rate rises least during the test, it almost invariably falls to a less extent after the test. But perhaps the strongest argument in favour of this view is the result of suddenly releasing a tight abdominal bandage at the moment of cessation of the test. Now the blood under pressure in the abdomen which normally floods to the heart is accumulated largely in the abdomen. Correspondingly we find that the pulse rate after the tests keep well above normal and the systolic blood pressure below normal—in other words, the usual effects are completely reversed.

Finally, it will be of value to consider the effects of various other factors on the results of the test. We find that stagnation of a considerable quantity of blood in the vessels of the skin (which involves compensatory splanchnic constriction) has but a small influence on the results of the test. This is shown in Table V. Probably a very much larger volume of

TABLE V.  
PULSE RECORDS OF SUBJECT W. D. H. (beats per ten seconds).

Graphs.	Average before test.	Average during test.	Average of highest values reached.	Recovery.	Average after test.	Lowest values reached.	Average of experiments.
23.1.22.							
25, i & ii. Normal test	11.0	11.6	13.2	12.0	10.8	9.6	2
26, i & ii. At 23° C. in stoke-hole	11.1	14.2	16.0	15.2	10.5	9.7	2
26, iii & iv. Normals after rest, at 14° C.	11.3	15.3	17.8	17.7	11.4	10.4	2
24.1.22.							
28, i & ii. In snow	10.8	13.1	14.9	13.9	10.1	8.9	2
28, iii & iv. In stoke-hole at 33° C.	11.4	13.8	15.4	15.3	10.9	9.6	2
28, v & vi. In snow again	9.2	10.8	12.4	12.0	9.6	8.9	2

blood is accumulated in the vessels of the muscles when the subject rests after muscular work. Table VI shows that after twenty minutes' stair-climbing the rise in pulse rate during the test is of the same degree as before the exercise, but that after the test a much greater average slowing takes place. (This agrees with the results of Hambly and McSwiney.<sup>3</sup>) This is possibly because more blood has been diverted to the dilated

limb vessels and has helped to flood the heart when the mercury is released. It will be noticed how the lowest values recorded after the test are the same in each case in Table VI, this probably representing the limit to which the vagus can slow the heart rate in this subject.

TABLE VI.

PULSE RECORDS ON SUBJECT W. D. H. (in beats per ten seconds).

Graph.	Average before test.	Average during test.	Highest values reached.	Recovery.	Average after test.	Lowest values.	Average of experiments.
23, i & vii. Normal.	10.9	13.7	15.5	15.5	10.7	9.5	2
Then twenty minutes' stair-climbing and test repeated at intervals afterwards.							
24, i. 2 mins. after exercise . . .	14.9	17.6	19.6	19.6	13.2	9.7	1
24, ii. 5 mins. after exercise . . .	14.1	16.7	19.2	18.1	12.7	9.3	1
24, iii. 10 mins. after exercise . . .	12.5	14.9	17.3	15.7	11.6	9.4	1
24, iv. 30 mins. after exercise . . .	11.7	14.0	18.5	14.0	11.2	9.3	1
24, v. 30 mins. after exercise . . .	11.6	13.9	17.2	13.5	11.3	9.6	1

Again, a few observations were made as to the effect of abdominal or thoracic breathing on subject C. A. N. H., who had remarkable control over his abdominal and thoracic muscles. We found that when he kept his abdominal muscles firmly contracted before, during and after the test, and raised the mercury entirely by contracting his thorax, his pulse rate rose ten beats per minute higher and his systolic blood pressure fell 10 mm. lower during the period of holding the mercury than when this was performed almost entirely by contracting his abdomen. The pulse and blood pressure records were almost identical in the period following the test. It appears, therefore, that as far as the results obtained during the test are concerned, the abdominal breather is in a better position than is the thoracic breather, but in the absence of comparative values of the intra-abdominal and intra-thoracic pressures, an explanation of this is difficult.

Again, we have arranged all our adult subjects in order of their physical fitness as judged by several different standards (Table VII). It will be seen that the least variations in pulse rate and in systolic blood pressure both during and after the test occur in the fitter subjects, and although this seems to indicate that the pulse response to the test (especially *during*

the test) may be of value as an indicator of physical fitness, we refrain from drawing any very definite conclusions from such a small number of subjects. (In the two subjects of fifteen and sixteen years of age respectively, our results of normal tests showed such extreme variations that we had to neglect results from the subjects.)

TABLE VII.

(Most fit subject placed first and others placed in sequence.)

Subject.	Resting pulse rate (per ten seconds).	Average during rest.	Rise.	Average after test.	Fall below normal.	Number of experiments.
F. C. S.	13.2	13.4	+ 0.2	12.2	- 1.0	4
R. F. C.*	15.3	20.7	+ 5.4	15.7	+ 0.4	2
A. B.	13.5	15.4	+ 1.9	12.6	- 0.9	3
W. W. P.	12.1	—	—	11.1	- 1.0	3
W. D. H.	11.0	13.5	+ 2.5	10.4	- 0.6	5
C. A. N. H.	11.8	15.2	+ 3.4	10.5	- 1.3	22
C. M.	14.8	19.8	+ 5.0	12.4	- 2.4	10

## DISCUSSION OF RESULTS

The above results show that the chief factor in causing the sudden fall in systolic blood pressure and rise in pulse rate at the commencement of the period of holding up the mercury is the collapse of the abdominal and thoracic veins under the external pressure to which they are subjected. The blood gradually accumulates on the arterial side of these vessels and finally renders them patent again. Thereafter the blood can again reach the heart and the systolic blood pressure rises and the pulse rate falls in accordance with Marey's Law. These results obtained during the test are in agreement with Flack's results,<sup>6</sup> but although he took pulse records throughout the period of the test, he appears to have overlooked the preliminary fall in systolic blood pressure (which is the key to our interpretation of the results), for the first readings he mentions are taken fifteen to twenty seconds after the commencement of the test, when as in our results the systolic blood pressure is rising again. It is to this that we attribute the divergence in conclusions which are drawn from the two sets of observations.

The subnormal phase of pulse rate which occurs after the mercury is released and to which Flack refers<sup>1</sup> is to be attributed to the sudden flooding of the heart with blood which occurs when the external pressure on the large veins of the thorax and abdomen is removed. To prevent an excessive rise of

\* This subject was profoundly affected by the psychological factor of the test, and nearly fainted.

blood pressure, the vagus comes into action and slows the heart rate.

Finally, we agree with Flack on the importance of good tone in both the abdominal wall and the abdominal vessels, and also that the more unfit subjects are those that show the greatest variations in pulse rate and in systolic blood pressure during and after the test. These variations we believe to be due to the much greater accumulation of blood in the splanchnic vessels (owing to the poor abdominal tone) which will take place before the compressed vessels are reopened.

In conclusion we would like to express our very best thanks to Professor M. S. Pembrey for his kind help and criticism which have been so valuable, to Dr. J. M. H. Campbell and Mr. W. W. Payne for help with x-rays and tests on the intra-œsophageal and intra-gastric pressures, and finally to Messrs. Baker, Chambers, Hicks, Moykopf and Smith who have so kindly acted as our subjects.

A preliminary account of this paper was read before the Physiological Society in October 1922.

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# THE ÆTIOLOGY AND PROGNOSIS OF ACUTE NEPHRITIS IN CHILDREN AND YOUNG ADULTS

## I.

### A CLINICAL STUDY OF FIFTY-SIX CASES

By A. A. OSMAN, D.S.C., Medical Assistant, Guy's Hospital.

THE object of this investigation was to determine the after results in a series of children and young adults, who had been admitted into the hospital with acute primary nephritis. The opportunity was also taken of making some inquiries into the possible causes of this condition. An attempt was made to follow up all such cases in the twenty-year period from 1900 to 1920 inclusive. Fifty-six cases were traced and attended for examination on two occasions each at an interval of from two to three months. The period which had elapsed since the onset of the illness to the date of the examination, of course, varied considerably; the shortest interval being eighteen months and the longest twenty-two years. The actual intervals and the number of cases for each are given here :

Onset of illness to date of examination in years . . . . .	1½	2	3½	4	4½	5	5½	6	6½	7	7½	8	9	9½	10	18	19	22
No. of cases . . . . .	4	6	1	2	2	7	8	5	2	5	4	3	1	2	1	1	1	1

Each case was submitted to an ordinary clinical examination, special attention being paid to the cardio-vascular system and the blood pressure. The urine was examined for albumen, and microscopically for blood, pus and casts. No elaborate chemical investigations of either the urine or the blood were undertaken, and none of the usual tests of renal function was performed. The whole research was entirely clinical. Very careful inquiry was made in each case into the individual response to those factors which are of such special importance in the daily life of a "nephritic," viz.: diet, climate, infection and exercise. Without in the least under-estimating the value of the well-known tests for renal efficiency, I believe that a very fair estimate of renal capacity can be formed in most cases by an intelligent inquiry into the response to the ordinary affairs of daily life, aided by a careful clinical examination.

## ÆTIOLOGY

It is generally agreed that most acute nephritis is infective in origin, though the point of entrance of the infecting organism

is often difficult to determine. In the present series of fifty-six cases the original illness appeared to have followed some acute inflammatory process in the upper respiratory tract, usually the tonsils, middle ear or both. This appeared to be the case in thirty-three, or 59 per cent. of the cases. In three instances the disease seemed to result from exposure and chill, without any history of local infection of the throat or elsewhere. Three cases resulted from pneumonia, and two were associated with purpura. In the remaining fifteen cases no reliable history of the onset could be obtained. In a very high proportion of the cases, therefore, the disease almost certainly resulted from infection of the upper respiratory tract, generally the tonsils.

In a further series of 235 patients, including both children and adults, the proportion of cases known to have followed infection of the throat was not so high, but, as none of these cases could be traced, and the figures were taken from the old case-histories, many of them defective, little reliance could be placed on them, and they are only mentioned here because the more unusual modes of onset were recorded and are of some interest.

Of the 235 cases, all in a first attack, twenty-eight died from the following causes :

Acute nephritis and suppression	10
Pneumonia	8
Septicæmia	3
Pleurisy	1
Probably acute on chronic nephritis	6

The probable predisposing causes were as follows :

No cause assignable (defective report)	69
Acute tonsillitis	86=36.5 per cent.
Lobar pneumonia	20
Sepsis (skin and scalp wounds)	11
Scarlet fever (contact)	10
Exposure and cold	8
Catarrh (common cold)	8
Empyema	4
Pleurisy	2
Acute rheumatism	4
Purpura	3
Syphilis (secondary)	1
Urotropine (gonorrhœa)	2
(Pregnancy)	(2)
?Previous nephritis	9
?Chronic nephritis	6



The list agrees well with the causes usually given in text-books. This series will not be referred to again here.

Of the fifty-six cases in the series forming the subject of this paper, only seven were known to have suffered from scarlet fever at any time previously, and in none of these did the nephritis follow directly upon the fever. This point is of interest, because, owing to the fact that scarlatinal nephritis figures in most text-books as the classical variety of acute glomerular nephritis both clinically and pathologically, the impression has arisen that the most frequent cause of acute nephritis in children is scarlet fever. Such an impression is entirely wrong.\* By far the most common cause of acute nephritis in children is infection of the throat and upper air passages. Incidentally it would be instructive to know how far removal of tonsils and attention to the hygiene of the throat generally would influence both incidence and prognosis. This aspect of the question is at present under investigation, but sufficient figures have not yet been obtained to include here.

#### REMOTE PROGNOSIS

The general opinion prevails that acute nephritis in children seldom results in chronic disease of the kidney. Further experience has shown that such a sequel is not very uncommon, and it is now recognised that even after an interval of many years unmistakable signs of chronic nephritis may supervene in a proportion of the cases. The results obtained in the present investigation support this view. Of the fifty-six cases, as judged by the methods already stated, thirty-six, or 64·2 per cent. appeared to be perfectly normal in every way. The remaining twenty, or 35·7 per cent., showed some signs of impaired renal function, which, considering the age of most of the cases and the absence of any other pertinent factor in the history, might fairly be attributed to the original nephritis. These twenty cases of incomplete recovery fall naturally into four well-defined groups. Before describing each group in turn, it may be stated that no definite correlation could be found between the ultimate condition of a case and the treatment adopted at the time of the original illness. Such factors as the severity of the initial illness, the length of time kept in bed, the diet, and whether or not discharged entirely albumen-free, did not appear to influence materially the ultimate result. It is probable, however, that similar observations on a much larger series of cases would reveal some such correlation.

\* Except in so far as scarlet fever itself is probably the result of an infection which enters the body through the tonsils or neighbouring parts. The incidence of nephritis in scarlet fever varies in different epidemics, but is usually given as from 6-11 per cent. of all cases.

*Group 1. Chronic Subacute Nephritis (Table I)*

In this group there were six cases. This type is characterised by more or less permanent ill-health. Two cases showed slight cardiac hypertrophy and a slightly raised systolic blood pressure. All had measurable amounts of albumen in the urine, from  $\frac{1}{4}$  to  $3\frac{1}{2}$  parts per 1000, and varying numbers of casts. They were all regarded as delicate individuals, with "weak" kidneys, and with one exception all stated that they were subject to recurrences of the old trouble from time to time. The exacerbations were generally attributed to colds and sore throats, but in one case invariably followed undue physical exertion. The attacks consisted of malaise, œdema, and blood in the urine, lasting from a few days only to many weeks. In this group the damage is permanent, but not necessarily progressive over the periods considered, though probably ultimately progressive. The damage is manifested partly by parenchymatous, and partly by interstitial and cardio-vascular changes. The efficiency of the kidney is permanently impaired, and, as in all the other groups, except 3, its resistance to infection and other deleterious influences is lowered.

*Group 2. Chronic, Intermittent, Hæmorrhagic Nephritis*

This unwieldy title has been employed to describe a form of acute nephritis, which is by no means rare in children. It is sufficiently unlike other varieties to warrant a separate description. One case only occurred in this series, but several further examples have been met with since, and they will be described elsewhere. A brief history of one case will serve to indicate the salient features of the type.

Stella A., aged 14, contracted a sore throat at the age of nine. The following day she complained of headache and nausea, and passed bright red urine. She was admitted to hospital with considerable œdema of the face, arms and legs, and slight ascites. The urine was bright red, contained seven parts per 1000 of albumen, but only a few casts (the type of cast was not stated). On a milk diet the œdema cleared up in about four days, and the urine became lighter in colour, and later free from blood to the naked eye. She remained in hospital just over six weeks, and was then discharged, passing a definite trace of albumen and a few red blood corpuscles, but otherwise quite well. During the next five years she had regularly two attacks of hæmaturia yearly. Each attack consisted of malaise and hæmaturia, but no œdema, and lasted about a fortnight or three weeks. Each attack was attributed to a cold in the head, sometimes with, and sometimes without, an accompanying sore

throat. During the intervals between the attacks the urine has been frequently examined, and was always normal. She is now a well-built, healthy-looking girl of fourteen years. There are no signs of cardiac hypertrophy, and her systolic blood pressure is 120 mm. of Hg. The urine is normal. Wagner, quoted by Pfaundler and Schlossman in their *Diseases and Disorders of Children*, describes a similar type of nephritis, under the title of chronic hæmorrhagic nephritis, and says that it is especially associated with tonsillitis, and, according to him, is characterised by absence of œdema, by acute exacerbations with scanty urine and little blood, and it has a good prognosis. These cases are probably closely related to those described during the war as associated with infected wounds. It appears to be almost an axiom that in acute nephritis, the more blood the better the outlook, both immediate and remote.

#### *Group 3. Leaky Kidney (Table III)*

There were three examples of this type. "Leaky Kidney" is a well-recognised sequel to acute nephritis. It is characterised by persistent albuminuria, but no casts, and no signs or symptoms of nephritis. The outlook appears to be entirely favourable.

#### *Group 4. Chronic Interstitial Nephritis (Table IV)*

In this group there were five cases. It is perhaps an exaggeration to diagnose chronic interstitial nephritis here. There was in each case a slight displacement of the apex beat, an increase of the aortic sound, and a slightly raised blood pressure (see Table IV). The urine was normal. Perhaps it would be more accurate to regard this condition as a transition stage between acute nephritis and chronic interstitial nephritis. The original illness in all was typical of acute nephritis. This sequence has been demonstrated in some cases of war nephritis, and if the cases described here can be followed up in the future, a similar relation may be shown to hold good in the nephritis of children.

No evidence bearing on the relation between acute diffuse nephritis and chronic parenchymatous nephritis was obtained from a study of this series.

### CONCLUSIONS

1. Fifty-six cases of acute nephritis in children have been followed up for varying periods of from 1½ to 22 years.
2. At least 55 per cent. of the cases are caused by infection of the upper respiratory tract, generally the tonsils.

Sex.	Age.	Probable Cause.	Condition on Admission.			Condition of Urine on Discharge.		Interval in Illness and Discharge.	Condition when Examined.			Age.	Previous Sore-throat Fever.
			Oedema.	Albumen.	Blood.	Albumen.	Blood.		Blood.	Albumen.	Casts.		
F	25 yrs.	Tonsillitis	Slight		8 pts. per 1000	++	+	5	Nil	+	+	29	Unknown
F	6½	?	Slight		2	++	Not reported	5	Nil	+	+	11½	No
F	3½	?	Very slight		1	++	+	5½	Nil	1½	+	8½	No
M	5½	Tonsillitis	Very slight		½	+++	Trace	6½	Nil	Trace	+	12	4 yrs. after onset
M	24	Tonsillitis	Marked		4	++	2	7	Nil	3½	+	32	Unknown
F	4	Tonsillitis	Not reported		2	++	Normal	7½	Nil	+	+	12	No

TABLE II.—*Chronic Intermittent Haemorrhagic Nephritis.*

F	9	Tonsillitis	Marked	9	+++	+	+	5	Normal			120	14	Yes
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TABLE III.—*Leaky Kidney.*

F	4½	?	Slight	1	+	Trace	Nil	5½	Nil	1	Nil	94	10	No
F	6½	?	Slight	½	++	Normal		7	Nil	½	Nil	112	14	Yes
F	2	?	Very slight	½	++	Normal		19½	Nil	½	Nil	124	21½	No

TABLE IV.—*Interstitial Nephritis.*

F	32	Tonsillitis	Moderate	4	++	Normal		2½	Normal			148	34½	No
M	16	Exposure	Marked	4	+	Not reported		2	Normal			130	18	No
F	14	Tonsillitis	Marked	1	++	Normal		5	Normal			132	19	No
M	13	Otitis	Marked	1½	++	Normal		6	Nil	Trace	Nil	132	19	No
M	15½	?	Marked	5	++	Trace	Nil	7½	Normal			144	22	Unknown

3. In those cases which survive no relation can be established between the severity of the original illness or the treatment adopted and the ultimate outlook.

4. A rather higher proportion of cases than expected (35·7 per cent.) showed signs of incomplete recovery.

5. Cases of incomplete recovery fall into four well-recognised groups.

6. No evidence bearing upon the relation between acute nephritis and chronic parenchymatous nephritis was obtained in this investigation.

## EOSINOPHILIA IN ECHINOCOCCUS INFECTION

By A. C. HAMPSON, Assistant House Surgeon, Guy's Hospital.

THE following case is chiefly remarkable for the high degree of eosinophilia, associated with hydatid disease of the liver.

R. J. was sent to Guy's Hospital by Dr. A. F. Hurst, and admitted on November 8, 1924. There was three months' history of a dull aching pain, which at the commencement of the trouble was located just above the umbilicus: it then occurred usually about 4 a.m. and lasted about half an hour. During the next two months the duration of the pain became gradually longer, till at the end of that period it was fairly constantly present; the pain was then chiefly located in the epigastrium. For the next two weeks the patient was dieted with considerable relief of pain, which, however, recurred at the end of that time and had been constantly present up to the time of admission. There had been no vomiting or constipation. For two days there had been an urticarial rash, which was still present on admission. On examination, an obvious elastic tumour could be seen in the epigastrium, bulging forward on either side of the rectus abdominis. An x-ray examination showed a shadow extending from the liver, on the right side to the level of the umbilicus, and on the left extending down to the left iliac fossa. An examination of the blood showed a leucocytosis of 16,500, of which 68 per cent. were eosinophils (absolute number of eosinophils per c.mm. = 11,200). A diagnosis of hydatid disease of the liver was made though no obvious source of the disease could be traced, and the patient had never been abroad. The patient was remarkably fit, and complained of nothing except a dull aching pain. Mr. L. Bromley operated on the evening of admission, and a large hydatid cyst was removed from the liver, which was stretched over the cyst, forming a very thin wall. The wound healed well, and the patient made an uneventful recovery, being discharged on November 27, 1924.

### *The Blood Picture*

The first examination of the blood was made on November 8, 1924, at 5.30 p.m., half an hour before operation. Subsequent examinations were made on each occasion at 5.30 p.m. The

number of white cells, and the differential counts are shown in the table.

Date.	Total White Cells.	Neutrophil Polymorphs.	Eosinophils.	Lymphocytes.	Hyalines.
Nov. 8	16,500	20 <sup>0</sup> / <sub>0</sub>	68 <sup>0</sup> / <sub>0</sub>	10 <sup>0</sup> / <sub>0</sub>	2 <sup>0</sup> / <sub>0</sub>
" 9	15,300	37 <sup>0</sup> / <sub>0</sub>	53 <sup>0</sup> / <sub>0</sub>	8 <sup>0</sup> / <sub>0</sub>	2 <sup>0</sup> / <sub>0</sub>
" 10	13,300	33 <sup>0</sup> / <sub>0</sub>	56 <sup>0</sup> / <sub>0</sub>	10 <sup>0</sup> / <sub>0</sub>	1 <sup>0</sup> / <sub>0</sub>
" 11	13,400	47 <sup>0</sup> / <sub>0</sub>	48 <sup>0</sup> / <sub>0</sub>	12 <sup>0</sup> / <sub>0</sub>	3 <sup>0</sup> / <sub>0</sub>
" 12	11,900	42 <sup>0</sup> / <sub>0</sub>	43 <sup>0</sup> / <sub>0</sub>	13 <sup>0</sup> / <sub>0</sub>	2 <sup>0</sup> / <sub>0</sub>
" 14	10,940	46 <sup>0</sup> / <sub>0</sub>	37 <sup>0</sup> / <sub>0</sub>	14 <sup>0</sup> / <sub>0</sub>	3 <sup>0</sup> / <sub>0</sub>
" 17	8,050	48 <sup>0</sup> / <sub>0</sub>	33 <sup>0</sup> / <sub>0</sub>	16 <sup>0</sup> / <sub>0</sub>	3 <sup>0</sup> / <sub>0</sub>
" 20	7,000	56 <sup>0</sup> / <sub>0</sub>	21 <sup>0</sup> / <sub>0</sub>	19 <sup>0</sup> / <sub>0</sub>	4 <sup>0</sup> / <sub>0</sub>
" 26	6,500	60 <sup>0</sup> / <sub>0</sub>	9.5 <sup>0</sup> / <sub>0</sub>	26.5 <sup>0</sup> / <sub>0</sub>	4 <sup>0</sup> / <sub>0</sub>
April 28	—	65 <sup>0</sup> / <sub>0</sub>	1 <sup>0</sup> / <sub>0</sub>	30 <sup>0</sup> / <sub>0</sub>	4 <sup>0</sup> / <sub>0</sub>

The absolute values expressed as the number of cells of each type per c.mm. of blood are shown in the figure below.

It will be noticed that the value of the hyalines and lymphocytes remained very constant. The value of the neutrophil polymorphonuclear cells showed a slight rise after the operation till November 17; this corresponded exactly with a slight rise of temperature during the same period. The values for neutrophils, lymphocytes and hyaline cells would correspond to a normal white count of about 5,800. The whole of the leucocytosis would therefore appear to be due to eosinophils, the curve for which descends almost parallel with that for the total number of white cells.

I have been unable to trace in the literature on this subject an eosinophilia of this degree occurring in hydatid disease, though several high figures are recorded; for example, Wagner<sup>1</sup> cites a case with eosinophilia of 64 per cent. with a leucocytosis of 13,000 (absolute number of eosinophils = 8,300) in a case of a ruptured cyst; and Augier<sup>2</sup> eosinophilia of 57 per cent. falling to 1 per cent. after operation.

An interesting feature was shown by comparison of the blood films before operation and twenty-four hours later. In the former the eosinophil cells were filled with granules; in the latter very few cells of this nature were found, the majority showing only a few granules set in a hyaline cytoplasm, which showed all degrees of vacuolation. The neutrophil polymorph cells were apparently unaffected. During the following days more and more cells showed an increased number of granules, till a week following the operation the eosinophil cells were similar to those before operation. This would suggest that the granules of these cells were used to protect against the proteins which were presumably thrown into the system at

the time of the operation. If this be so, it would seem that the stimulus of the small protein leakage before operation produced an eosinophilia more than sufficient to protect against

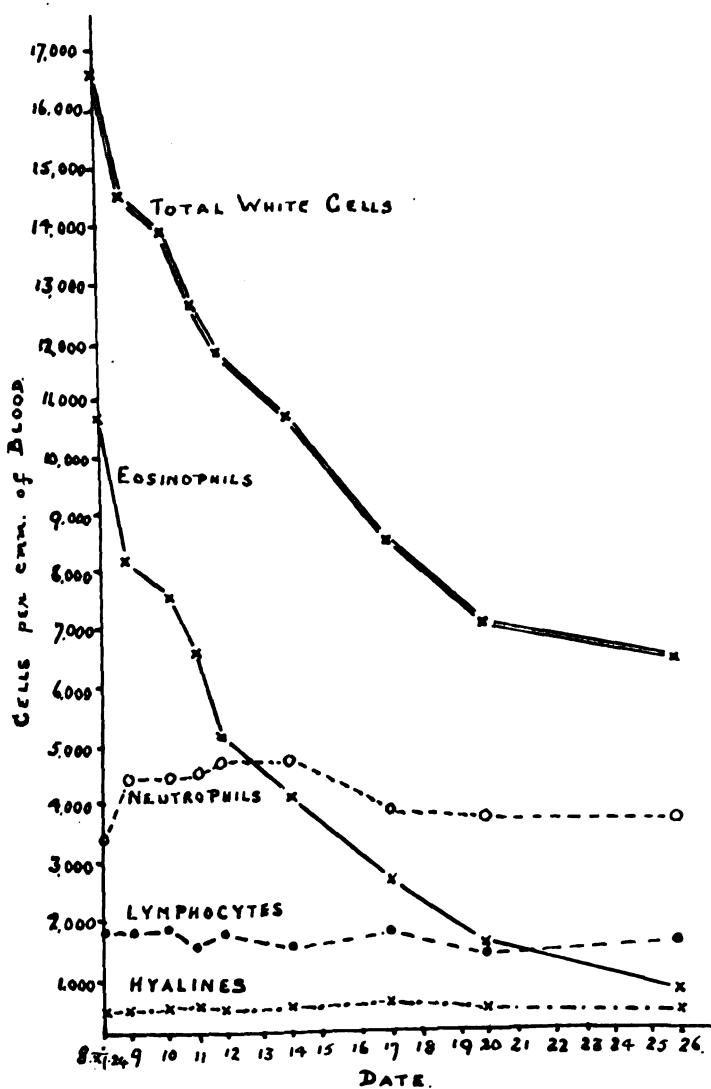


FIG. 1.

it—in fact, the over-reaction to a stimulus so commonly found in physiological reactions. This is of interest also in connection with the controversy as to the efficacy of artificially produced polymorphonuclear leucocytosis (e.g. by administration of nuclein) in cases of sepsis.



I should like to express my sincere thanks to Dr. A. F. Hurst and Mr. L. Bromley for permission to publish this case.

## REFERENCES

- <sup>1</sup> K. E. Wagner : *Zen'tralbl. f. inn. Med.*, xxix. 129, 1908.
- <sup>2</sup> M. Augier : *Fol. Haematol.*, ix. 128, 1910.

## A CASE OF TETANUS

By FRANK MARSH, Assistant Bacteriologist, and R. B. FAWKES, D.S.O.,  
B.Ch., House Physician, Guy's Hospital.

On the 17th of April, 1925, a man of 41 years of age was admitted to Guy's Hospital under Dr. Marshall suffering from tetanus.

### *Clinical History*

On April 10th he trod on a three-inch rusty nail lying in the manure from a fowl-house in his garden. This caused a deep punctured wound between the heads of the 3rd and 4th metatarsal bones of his left foot; there was very slight bleeding. He bathed his foot in hot water. Next morning his foot ached badly, so he called in his doctor, who ordered hot fomentations. The wound healed.

On April 17th at 2 p.m. patient noticed that his jaws were slightly stiff. At 8 p.m. that day he was admitted to Guy's Hospital, by which time the maximum separation between upper and lower incisors was  $\frac{1}{4}$ ".

The original puncture had healed, and there was no obvious local rigidity. Fibrillary twitching of masseters and early *risus sardonicus* were present. There was some limitation of forward movement of the head and of forward bending of the spine, with some rigidity of the recti abdominales. The mental state and reflexes were normal.

Dr. Marshall advised confining anti-tetanus serum to intramuscular and intravenous routes, on the ground that statistics of the war cases showed better results when intrathecal administration was omitted, and that he had seen a fatal case of aseptic meningitis following intrathecal administration of anti-tetanus serum. At 9 p.m. anti-tetanus serum was administered, 1,500 units locally round wound, 3,000 intramuscularly into the quadriceps femoris, and 16,000 intravenously. The administration of serum was followed by a rise of temperature and vomiting.

At 10 p.m. the site of the healed nail track was excised throughout its length, half-an-inch of tissue being included all round the wound. Most of the trismus disappeared while the patient was under the anæsthetic. The next day, April 18th, he was given 48,000 units of anti-tetanus serum,—8,000 intramuscularly, and 40,000 intravenously. Slight spasms now occurred in all the muscles previously showing rigidity. Morphia was administered.

On the 19th the spasms became more severe and were accompanied by cyanosis. 48,000 units anti-tetanus serum were given as before, and bromide was administered.

On the 20th there was slight improvement, relaxation of the jaw being noticeable early in the day. Later severe spasms occurred with much cyanosis, and paresis of the left half of diaphragm developed. Swallowing became impossible, and rectal medication and feeding were resorted to. 48,000 anti-tetanus serum were given as before.

On the 21st light chloroform anæsthesia was employed during spasms. Continuous oxygen inhalation failed to relieve the rapidly increasing broncho-pneumonia. 48,000 units anti-tetanus serum were given as before.

Death occurred at 3 a.m. on April 22nd.

Total serum administered :—Units 212,000.

Locally . . . . .	units 1,500
Intramuscularly . . . . .	units 35,000
Intravenously . . . . .	units 176,000

#### *Bacteriological Examination*

The mass of tissue excised at operation was dropped into a sterile specimen tube, the tissue covered with glucose broth and the tube closed with a sterile cork.

An agar slope and also a blood agar slope were infected with a swab from the excision wound. The three tubes were then incubated anaerobically in a Laidlaw's jar at 37° C.

On examination after forty-eight hours a large Gram-positive bacillus with a sub-terminal spore very little thicker than the body of the organism was seen in all the cultures, apparently unmixed with any other organism.

On examination after three days, the glucose broth culture exhibited a strong odour of putrefaction, and the fragment of tissue had been disintegrated into portions the size of a match head.

Microscopically a bacillus morphologically identical with *B. tetani*, complete with large round terminal spores, was seen in considerable numbers, mingled with the bacillus originally observed.

The cultures on solid media grew the contaminating organism only.

The tissue-containing tube was heated in a water bath at 80° C. for fifteen minutes. Then three tubes of Robertson's bullock's heart medium were infected from the heated culture and incubated in Laidlaw's jar. After forty-eight hours *B.*

*tetani*-like forms mixed with the contaminating bacillus were still present. It was obvious that the heating method alone was useless.

Another attempt to obtain a pure culture of *B. tetani* was made by Sturges and Rettger's method.<sup>1</sup> Loopfuls of the meat media were mixed with *Staphylococcus aureus* and spread on agar plates and incubated aerobically. Only *Staphylococcus aureus* and the contaminating bacillus grew.

Fildes' method was next tried.<sup>2</sup> The condensation water

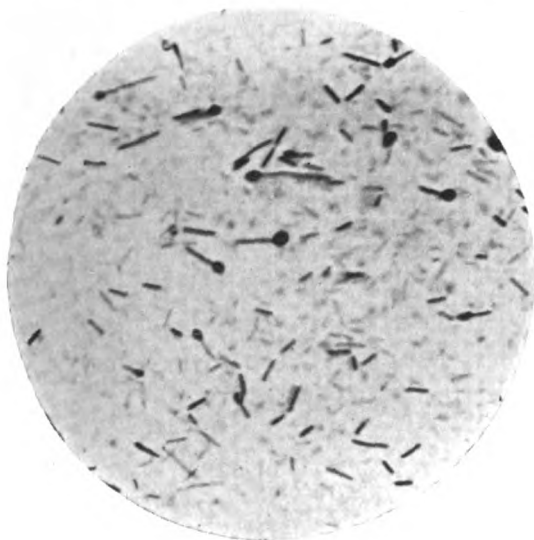


FIG. 1.

Film preparation from transparent growth at upper part of agar slant culture after incubation at 37°C. anaerobically for 4 days  $\times 1100$ .

of four agar tubes was infected from the meat tubes and incubated anaerobically. At twenty-four hours spikes of growth were seen climbing up the surface of the slopes. The surface of the agar was hopefully scraped with a platinum loop above the highest point of visible growth, but no microbes could be seen in the scrapings. The visible growth proved to be the contaminating bacillus alone.

Several further trials of this method were made and finally complete success was attained after an incubation period of four days. A thin, transparent, moist growth appeared at the upper end of the slope which on microscopical examination proved to be the *B. tetanus*-like form alone (Fig. 1).

Having obtained the organism in pure culture, the next

step was to determine whether it was capable of producing a toxin comparable to that of *B. tetani*.

Two fresh meat tubes were infected and incubated anaerobically at 37° C. for seven days. At the end of that time the contents of the two tubes mixed with 10 c.c. distilled water were filtered through a candle. Two c.c. of the filtrate were injected into a mouse, 1 c.c. at the root of its tail and 1 c.c. under the skin of its abdomen. The mouse was watched continuously for half-an-hour, but beyond slight paresis of its hind legs it seemed none the worse.

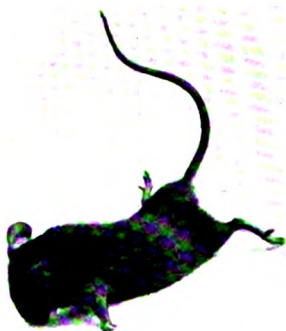


FIG. 2.

Cadaver of mouse which was found dead with marked opisthotonos 10 hours after subcutaneous injection of 2 c.c. filtrate from a 7-day broth culture.

Ten hours later the mouse was found dead in its box in marked opisthotonos (Fig. 2).

It was thought advisable to publish the case for the purpose of emphasising the following points.

(1) The unusual fact of the isolation of *B. tetani* from a healed wound.

(2) The virulence of the infection as indicated by the rapid onset of tetanic symptoms (just inside seven days), and the consonance of the fatal result with the usual experience of cases with so short an incubation period.

(3) The inadequacy of *curative* treatment (particularly when intrathecal injections are omitted) as compared with the consistently good results of *preventive* treatment.

(4) The danger of disregarding the lessons of the war concerning the prophylactic use of anti-tetanus serum.

Our thanks are due to Dr. Beddard and Dr. Marshall for granting us permission to publish this case. We have to express our deep indebtedness to Professor Eyre for his cordial encouragement and help in the bacteriological investigation, and in the preparation of this paper.

The two excellent photographs are due to Mr. A. Durrant, Laboratory Assistant.

#### REFERENCES

<sup>1</sup> Sturges and Rettger: *Journ. Bact.*, iv. 171, 1919.

<sup>2</sup> Fildes: *Brit. Journ. Exp. Path.*, vi. 62, 1925.

NOTE.—Since the above was written another case of tetanus with an incubation period of nineteen days was admitted to the same ward. The patient was injected intramuscularly and intravenously with anti-tetanic serum on admission and again intramuscularly, intravenously and intrathecally after being 48 hours in the ward, but he died 24 hours after this second administration of the serum with signs of severe meningeal irritation.

The quantity of anti-tetanic serum administered intrathecally was 8,000 units, contained in 10 c.c. of serum, 20 c.c. of cerebro-spinal fluid being withdrawn before injecting.

## OPERATION FOR ACUTE APPENDICITIS

By R. P. ROWLANDS, M.S., Surgeon to Guy's Hospital.

IN the last number of the Reports the writer drew attention to the disquieting fact that nearly 3000 deaths from appendicitis are reported annually in England and Wales. Most of these deaths (and many others incorrectly reported) are due to delay in treatment by the safest of all methods, *e.g.* early operation, but some appear to be due to errors of judgment and technic at the operation. Therefore it may be worth while offering some suggestions which may make the operation simpler, safer, and more successful.

The removal of the appendix in the early hours of the first acute attack of appendicitis is nearly always a simple, easy and safe matter in good hands, but previous attacks may have caused troublesome adhesions, making the operation a difficult one. Similarly, delayed operations, which have to be performed during an acute attack, may be very difficult and dangerous, except in skilled hands controlled by experience and sound judgment. In many cases opening and draining the abscess without doing any harm is all that can or ought to be done at this stage.

### *Choice of Incision*

Nearly all surgeons who have had a large experience of appendicitis choose the gridiron incision of McBurney. It affords a more direct approach than the Battle, rectus or paramedian incisions, and it allows drainage with far less risk of ventral hernia or damaging adhesions, which may subsequently cause intestinal obstruction, as shown by the clinical experience of many surgeons. It is commonly objected that the grid does not give enough room, and that it cannot be adequately enlarged for the treatment of disease in the pelvis or upper abdomen when a mistake in diagnosis has been made. I have never failed to find or remove the appendix through the grid, the access being made easier by invariably continuing the deep part of the incision inwards at least an inch into the rectus sheath and, if necessary, downwards or upwards for two or three inches from the inner end of the transverse incision in

the anterior wall of the rectus sheath. If the disease proves to be in the upper abdomen it is wise to make a separate incision higher up and directly over the seat of disease, the grid being closed in a few minutes or, in some cases, used for drainage. The Davis or Harrington modification of the grid, in which the external oblique fibres are cut across, is a very good one—especially for late cases requiring drainage—which can be easily carried out through the outer angle of the horizontal wound without risk of hernia.

Vertical incisions further inwards, towards the middle line, are over the small intestine instead of over the cæcum, and they disturb the intestines much more and often cause trouble from prolapse unless the anæsthesia is perfect: in my experience ventral hernia and intestinal obstruction are certainly more common after them. Moreover, they take more time to close adequately and securely.

The deep layers of the Battle incision, in which the rectus is displaced inwards, are notoriously difficult to close satisfactorily. Moreover, the nerve supply of the rectus may be damaged by its enlargement. For these reasons ventral hernia is not uncommon after this incision.

When I feel uncertain of the diagnosis I make a moderate right paramedian incision, displacing the rectus outwards and enlarging the wound either upwards or downwards if necessary.

### *Early Operation*

The grid incision, four or five inches long, is made a little lower than the lower point of trisection of the distance between the right anterior superior spine and the umbilicus. After separating the fibres of the external oblique for four inches and those of the deep flat muscles for three inches, opening the rectus sheath and drawing this muscle inwards, the incision in the parietal peritoneum is made only just large enough to allow the cæcum to be easily delivered and replaced, for this makes its subsequent closure easier. The cæcum is best sought and delivered by means of the two index fingers, and it is nearly always wise to deliver it, for this makes the removal of the appendix much easier and safer. In some late cases, however, adhesions hinder or prevent this step.

The appendix is best found by following the longitudinal bands of the cæcum downwards into it. Having separated any external adhesions that may be present around the appendix or its mesentery, the meso-appendix is pierced close to the cæcum and the root of the appendix, and a strong ligature is drawn through this perforation to tie the mesentery in one



piece, thus certainly securing all the blood vessels. The base of the appendix is clamped with two artery forceps in contact with each other. The meso-appendix is divided well beyond the ligature and a fine purse-string sero-muscular suture is introduced by a fine round needle, a quarter of an inch away from the base of the appendix, which is then tied, divided with a knife and invaginated by tying the purse-string. There is no need to use a special crushing forceps or carbolic acid for the base of the appendix. A coarse needle is apt to pierce the thin-walled cæcum and thus to contaminate the wound—in the past a common cause of abscess in clean cases. The two corners of the peritoneal wound are held and lifted by a toothed artery forceps and closed with a purse-string of fine catgut. The wound in the deep muscles is closed with a “figure of eight” suture, thus saving another knot and securing accurate apposition. The external oblique wound is closed with a double “figure of eight” suture, and the skin with a continuous suture of fine catgut or linen thread.

Carried out in a methodical way the operation can usually be well done and completed within ten or fifteen minutes, to the great advantage of the patient.

#### *Late Operation*

When a swelling is present the grid incision is placed over it and is sometimes longer than the usual four or five inches, to allow plenty of room for packing. Directly the peritoneum is opened and an abscess is felt a gauze roll is gently paid out to pack off the abscess from the general peritoneum, and the parietal wound is protected with pads. Then the abscess is opened with the finger, generally passed behind the lower part of the cæcum. All the pus is mopped away and, unless the abscess is late and very large and the patient ill, old or exhausted, the appendix is sought and removed without contaminating the general peritoneum or exposing or handling unduly any of the small intestine. When the appendix is very adherent it is a good plan to shell it out of its sero-muscular coat: this saves time, bleeding, and anxiety. The hidden, unknown leak of infective material is the greatest danger, and is best avoided by preliminary packing off and, later, by passing a small pad on a holder to the bottom of the pelvis, to prove the latter dry and clean. If any pus or blood is found there, it is all removed with a dry gauze roll passed into the pelvis and left in position for a few minutes.

The question of how, when, and where to drain is very important in these late cases, and the answer depends on the

local and general conditions. When I am satisfied that the peritoncum is dry and clean, especially as regards the pelvis, I do not drain: when, on the other hand, it is impossible to leave the pelvic peritoneum or right loin clean and dry, and especially if there has been a definite abscess of considerable size with persistent oozing of blood, I consider drainage essential, especially in old, enfeebled or exhausted patients. In late and desperate cases I am content merely to drain the abscess, and to leave the removal of the appendix to a safer and more favourable opportunity. About 25 per cent. of such cases suffer from recurrence of symptoms, sooner or later, unless the appendix is removed, and many a patient prefers not to run the risk, especially if he contemplates travelling, out of reach of expert surgery.

The greatest value of a drainage tube is in keeping open a gap in the abdominal wall as a vent. It is rarely necessary or advantageous for a tube to pass down to the bottom of the pelvis, or across coils of small intestine or important blood vessels, to cause pressure necrosis, with fæcal fistula, or secondary hæmorrhage. Therefore I insert a large rubber tube,  $\frac{1}{2}$  inch in diameter, just through the parietes and, in some cases of deep abscess, a piece of thin, soft rubber tubing (the size of a No. 10 catheter) is inserted through this tube, extending to near the site of the abscess. An inch of this inner tube is cut off every day.

As regards the position of the tube this depends on the site of the abscess. In most cases, with an abscess behind or near the cæcum, the tube is placed in the grid or at the outer angle of the Harrington incision. When the abscess is in the pelvis I place the tube in a stab wound near the middle line, close to the pubis but clear of the bladder, or in some cases, in women, through a wound carefully made from the pouch of Douglas into the vagina. Gauze should not be used for drainage because it soon acts as a plug, keeping in the discharges instead of draining them away; moreover, its early removal is so painful as to demand an anæsthetic. Folded rubber sheeting is much more efficient and humane.

In some very late cases paralytic distension complicates appendicitis. In such cases either primary or secondary valvular cæcostomy, passing the tube through the ileo-cæcal valve into the ileum, is a life-saving measure and, in some cases, a lateral anastomosis between the ileum or jejunum and the transverse colon, as advised by Handley, is valuable. In grave cases valvular jejunostomy is necessary to overcome paralytic distension.

Secondary abscesses, such as pelvic, lumbar or sub-diaphragmatic, may occur in neglected and late cases, but they are getting less common, in my experience, owing to earlier operation and more thorough packing off during operations on suppurative cases. Persistence of fever, leucocytosis and abdominal or pelvic distension generally indicate this complication. When the abscess has been definitely diagnosed it must be opened without delay after locating it either by clinical examination or laparotomy, but it is a mistake to re-explore the abdomen on chance or to give repeated anæsthetics without due cause, for these lower the resistance of the patient so much that they contribute very seriously to a fatal issue.

#### *After-Treatment*

Peritonitis, pulmonary embolism, and pulmonary complications are the chief causes of death after operations for acute appendicitis. They are almost entirely due to delay and can only be avoided by early operation. Thrombosis and pulmonary embolism are best avoided by early active and passive movements and by avoidance of pressure upon the vessels or limbs by tubes or knee pillows: also by the earliest possible return to full diet and the prevention of constipation.

#### *Mortality*

Apart from the mortality incidental to every anæsthetic and operation such as the radical cure of hernia, there is no risk in the early operation for acute appendicitis, carried out by a capable and aseptic surgeon. As regards the anæsthetic, warm ether with plenty of oxygen, given for less than half an hour, is almost devoid of danger. At the London Hospital<sup>1</sup> during the years 1920 to 1923 there were 221 operations undertaken within twenty-four hours of the onset of symptoms, with only two deaths, or 0·9 per cent. In a long experience I have not lost a single patient operated upon within twenty-four hours. It has been conclusively shown by many surgeons that the main cause of death is delay, therefore the operation should be carried out as soon as possible after the diagnosis is made or strongly suspected. In my wards at Guy's Hospital it has been the custom to operate upon all cases of acute appendicitis as soon as possible after admission and not to refuse or defer operation because of the lateness of the disease or the grave condition of the patient. In such cases measures are taken to resuscitate the patient (such as saline glucose infusions) and the operation is carried out without further delay. Mr. B. L. Laver, my present Surgical Registrar, found that between

1919 and 1925 there were, in my wards, 408 operations for acute appendicitis with 18 deaths, a mortality of 4·44 per cent. At autopsy ten had general peritonitis, three pneumonia, two pulmonary embolism and one empyema. The mortality of the 16 cases coming in with diffuse peritonitis was 25 per cent., whereas it was only 0·4 per cent. in the 223 cases where the disease was limited to the appendix, and 2·9 per cent. in 36 cases of abscess. There were no deaths in the 36 cases of localised peritonitis.

These results are good, especially when it is remembered that no case was refused operation, and that most of the patients admitted to Guy's Hospital come very late and from a poor district. Most of the operations were performed by my junior colleague, Mr. L. Bromley, and by my Surgical Registrars.

#### REFERENCE

- <sup>1</sup> R. J. McNeill Love : *Brit. Jour. Surg.*, xii. 232, 1924.

# MASSAGE AND REMEDIAL EXERCISES IN MEDICINE

## PART II. THE ABDOMINAL AND PELVIC MUSCLES, AND THEIR TREATMENT BY MASSAGE AND REMEDIAL EXERCISES IN VISCEROPTOSIS, RECTAL AND UTERINE PROLAPSE AND CONSTIPATION

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital.

### POSTURAL TONE AND CONTRACTILE POWER

IN any discussion on treatment by exercises it is essential to bear in mind that voluntary muscles, like involuntary muscles, have two distinct functions, tone and contractile power, either of which may be deficient independently of the other. As the form of physiotherapy which is useful for deficient contractile power has comparatively little effect on deficient tone, and *vice versa*, it is clear that disappointing results will be frequent unless the distinction between these two functions and their



respective treatment is clearly understood. So far as I know, however, no attempt has hitherto been made to distinguish between the treatment by exercises of deficient tone and that of deficient contractility.

The posture of an individual is maintained by what Sherrington<sup>1</sup> has called the postural tone of his muscles. The fingers, for example, are, when at rest, slightly flexed, and remain so not only in the waking state, but also during sleep and during anaesthesia. Each resting muscle has a certain degree of tone; and as for the greater part of every twenty-four hours it is at rest, its anatomical structure adapts itself to this tone; but it is capable of becoming shorter as a result of active contraction and longer as a result of active relaxation. If, for example, the line AB in Diagram I represents the length of a muscle in extreme relaxation and AC its length in extreme contraction, its normal

“postural length,” which depends upon its “postural tone,” will be AD. It is capable of shortening from AD to AC on active contraction, and lengthening from AD to AB on active relaxation. If as a result of continual stretching its postural length has been increased from AD to AD' (Diagram II), a greater effort is required to produce the maximal contraction CD' compared with the normal CD, but, however capable the muscle may be of doing this, the postural tone will remain unaltered and insufficient at AD'. On the other hand, with normal tone, AD, the contractile power of the muscle may be deficient, so that the muscle cannot be shortened beyond AC' (Diagram III). In practice, deficient postural tone is frequently present with normal contractile power, but, in the absence of organic disease, deficient contractile power is uncommon without some deficiency in postural tone.

It is necessary in discussing the question of tone to refer briefly to the recent work of John Irvine Hunter,<sup>2</sup> which suggests that voluntary muscles do not, as was formerly believed, consist of one kind of fibre only, having the double function of causing contraction and maintaining tone, but that they consist of two sets of fibres disposed in groups, each of which has its own specific innervation and function. In one set the fibres are large and are supplied by medullated spinal nerves ending in muscle plates; they are concerned in voluntary and reflex contraction, and in isometric contraction during the continuation of the stimulus, the latter constituting “contractile tone” (Langelaan). In the other set the fibres are slender and are supplied by non-medullated sympathetic nerves with “grape-like” terminations; these are concerned in the maintenance of “plastic tone” (Sherrington), being first inhibited and then shortened or lengthened to the extent necessary to maintain the new length imposed upon them by the contractile tone brought about by the activity of the larger fibres.

Both contractile and plastic tone depend upon the existence of reflexes, the different impulses of which originate in the muscles themselves. These two forms of tone together result in Sherrington's postural tone: contractile tone produces posture as a result of shortening of appropriate muscles; plastic tone of the fine fibres maintains this position.

#### THE ABDOMINAL MUSCLES IN DEFECATION

Owing to the weight of the abdominal viscera the pressure in the rectum is about 25 mm. of mercury, or approximately 15 mm. higher than the general intra-abdominal pressure, which

depends upon the normal postural tone of the diaphragm and abdominal and pelvic muscles. When the call to defæcation is felt on the arrival in the rectum of fæces from the pelvic colon, the act itself is voluntarily initiated by the simultaneous contraction of the diaphragm and abdominal muscles; this causes the intra-rectal pressure to rise to between 100 and 200 mm. of mercury (Keith).<sup>3</sup> The diminution in volume of the abdominal contents which results in the rise in pressure is brought about mainly by the contraction of the diaphragm. In 1906 E. G. Slesinger and I<sup>4</sup> watched with the x-rays the descent of the diaphragm during defæcation to the lowest position it could possibly attain, where, as a result of closure of the glottis, it remained with slight variations during the straining which resulted in the evacuation of the fæces.

The contraction of the abdominal muscles acts by offering a firm resistance and only to a comparatively slight extent by actually reducing the volume of the abdominal contents; in its absence the anterior abdominal wall would bulge and the effect of the descent of the diaphragm on the intra-abdominal pressure would be lost. If the contractile power of the abdominal muscles is unimpaired in fat people and individuals with deficient postural tone of these muscles, their contraction may greatly diminish the volume of the abdominal contents by replacing the convex protruding abdominal wall by a concave or flat wall.

The muscles of the pelvic floor do not take any active part in increasing the intra-abdominal pressure during defæcation, but as a result of their postural tone they offer an adequate resistance to pressure from above, and allow the diaphragm and abdominal muscles to create the necessary rise in pressure.

The involuntary contraction of the rectal wall caused by the reflex set up by the high pressure in its interior presses the fæces through the relaxed sphincters; the reflex is also directly assisted by the rise of the intra-abdominal pressure, which has the effect of extruding the fæces through the anal canal.

#### THE ABDOMINAL MUSCLES AND THE INTRA-ABDOMINAL PRESSURE

The viscera are normally kept in position by the slight positive intra-abdominal pressure, which measures about 10 mm. of mercury and is maintained by the postural tone of the muscles of the abdominal wall and the pelvic floor, their peritoneal attachments remaining slack. The position of the diaphragm also depends in part upon the difference between the negative intra-thoracic pressure and the positive intra-abdominal pres-

sure; if the latter falls for any reason, the diaphragm and the organs which are attached to it descend when the vertical position is assumed.

The efficiency of the circulation depends upon the maintenance of an adequate intra-abdominal pressure. Consequently deficient tone of the abdominal and pelvic muscles leads to an impaired circulation, and many of the symptoms commonly ascribed to the associated visceroptosis are really due to this.

Kelling <sup>5</sup> has demonstrated that a reflex mechanism exists, as a result of which the tone of the abdominal muscles adapts itself to the varying contents of the alimentary canal. In the absence of such a mechanism the consumption of a meal of moderate size, which increases the bulk of the abdominal contents by as much as 25 to 35 per cent., would cause a considerable rise of intra-abdominal and consequently of intra-gastric pressure, and the latter would cause an unpleasant sense of fullness.

The gradual stretching of the abdominal muscles during pregnancy is accompanied by some diminution in their postural tone. This does not, however, occur *pari passu* with the increasing volume of the abdominal contents, corresponding with what happens on eating a big meal, as Paramore <sup>6</sup> has shown that a considerable rise in intra-abdominal pressure occurs during pregnancy. Consequently after parturition the abdominal muscles, though very lax, are less so than would otherwise be the case. If the woman is kept in the recumbent position sufficiently long, so that no pressure is exerted on the abdominal muscles from within, they gradually regain their normal postural length and postural tone, and the separated recti come together. At the same time their contractile power is restored, and when the erect position is once more assumed the abdominal muscles are as efficient as ever, and neither dyschezia nor visceroptosis results.

#### THE ABDOMINAL MUSCLES IN DYSCHEZIA AND VISCEROPTOSIS

If insufficient rest is allowed after parturition, the weight of the viscera in the erect position keeps the abdominal muscles stretched; neither their tone nor contractile power returns to normal, and visceroptosis and dyschezia, or insufficient defæcation, result. The abdominal muscles are then inefficient in defæcation not only because of their diminished contractility, but also because their increased postural length necessitates a proportional increase in the force required to produce the necessary rise in intra-abdominal pressure.



The diminution in the bulk of the abdominal contents caused by loss of some of the intra-abdominal fat, when for any reason an individual loses weight, results in a fall of intra-abdominal pressure and consequently in visceroptosis, unless the postural tone of the abdominal muscles correspondingly increases. I have seen several cases in which visceroptosis, often accompanied by serious digestive symptoms, immediately followed the removal of a pelvic tumour, the intestines dropping into the position which was previously occupied by the latter; this can only be prevented by keeping the patient at rest for sufficiently long after the operation, and giving her exercises with the object of increasing the postural tone of the stretched abdominal muscles. The removal of ascites by paracentesis has a similar result; when the fluid collects rapidly, and especially if tapping is delayed, the sudden stretching of the abdominal muscles is very likely to produce permanent and serious loss in their efficiency.

Weakness of the abdominal muscles is common among people who take too little exercise owing to a sedentary occupation, laziness or obesity. The atrophy and weakness produced by want of exercise are often associated with a diminution in the postural tone, owing to the active relaxation of the abdominal muscles which occurs when an individual habitually slouches instead of walks and reclines in an easy chair instead of sitting up.

The malnutrition, which occurs in rickets, after acute fevers and in chronic infections, results in both weakness and loss of tone in the abdominal muscles. Under these various conditions dyschezia and visceroptosis result.

It is important to realise that the constipation, which is very frequently associated with visceroptosis, is not due to intestinal kinks, but is a result of dyschezia, both the inefficient defæcation and the visceroptosis being direct results of the weak abdominal muscles.

#### EXERCISES FOR THE DIAPHRAGM AND ABDOMINAL MUSCLES IN DYSCHESIA AND VISCEROPTOSIS

At first each movement may be repeated six times morning and evening, the number being gradually increased as the muscles become stronger, but the exact time to spend over the exercises varies in each case and depends upon the amount of fatigue produced, for the patient should always stop before he feels very tired. It is generally necessary to continue the

exercises for several months or even permanently, but after a time the number done can be slowly diminished.

It is important to remember that exercises are just as essential for the transversalis, internal and external oblique muscles as for the recti.

The following is a list of the most generally useful exercises, but they require modification to suit individual cases, and in most instances only a selection from them is likely to be required.

#### *A. Lying*

1. Slowly sit up with the arms stretched forwards until the finger-tips touch the feet; then slowly lie down again. When the muscles are very weak, it may be necessary at first to have the shoulders supported at the commencement of the movement and the feet held down.

2. Clasp the hands together behind the neck; raise the extended legs as high as possible and slowly let them fall. Again it is often necessary at first to have the legs supported.

3. Sharply draw in the abdomen, let it out again, and then push it out by contracting the diaphragm. When the condition of the muscles has improved, the exercise may be repeated with a sand-bag on the abdomen; the weight of the sand should be gradually increased from four to ten pounds.

4. With the arms at the sides and one foot laid over the other, raise the hips as far as possible from the ground, at first without resistance and later against the opposition of an assistant: then slowly lie down again.

#### *B. Sitting*

The patient sits with the feet firmly held on the ground and falls backwards from the hips, the back and neck remaining rigid; he then raises himself to the sitting position again.

#### *C. Standing*

1. Raise the arms above the head with inspiration; lower them to the sides with expiration, completing the movement with a voluntary contraction of the diaphragm.

2. Extend the arms above the head and keep the legs stiffly extended; bend the trunk forwards and try to touch the toes with the finger-tips.

3. Hang the arms by the side and keep the thighs and legs stiffly extended; bend first to one side and then to the other, trying to touch the foot with the hand of the same side.

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4. With the hands on the hips, twist the body round as far as possible, first in one direction and then in the other.

5. With the hands on the hips, which must remain fixed, lean back as far as possible, and slowly move the body so that the head and shoulders describe a large circle.

6. Raise the legs alternately as high as possible, so as to compress the abdomen with the thighs.

7. With the hands on the hips, stand on the toes and slowly bend the knees outwards with the body bent forwards, so as to assume a squatting position with the buttocks touching the heels and the thighs pressing on the abdomen; then slowly rise again.

### D. "*Walking on All Fours*"

In this exercise, the value of which was first emphasized by Léon Meunier,<sup>7</sup> the patient returns to the attitude of his ancestors, and restores his abdominal organs to the position they would have maintained had he never assumed the erect posture. When walking on all fours, the back leg is straightened, and then brought to a fully flexed position in which the abdomen is compressed by the muscles of the thigh. This exercise is remarkably efficacious in severe cases of visceroptosis.

### *Correct Posture*

When the postural tone of the abdominal muscles is deficient, great stress must be laid on the importance of adopting the correct posture when sitting, standing and walking. The shoulders must be held down and slightly back, the lumbar spine straightened and not hollowed, and the abdomen slightly contracted. When at meals, the patient should sit well back in his chair, with the head erect and the abdominal muscles firmly contracted. If for a few days the patient makes an effort to think of and to maintain a correct posture, by the voluntary production of a proper contractile tone of the large fibres of his abdominal and other muscles for several minutes at a time at frequent intervals, whatever else he may be doing, a new and improved plastic tone will soon be acquired by the fine muscle fibres so that no further voluntary effort will be required to maintain a correct posture.

### THE TREATMENT OF CONSTIPATION BY MASSAGE

Whenever constipation is due to want of activity of the intestinal musculature, the condition of the latter may be improved by abdominal massage, which exerts a directly

stimulating action upon it. This can be readily demonstrated in cases of organic obstruction, when peristalsis can often be seen after manipulating the abdomen. Case <sup>8</sup> has also shown with the x-rays that massage may give rise to mass peristalsis of the colon. In dyschezia the atonic and parietic pelvic colon and rectum cannot be influenced by massage owing to their situation in the pelvis, but massage is beneficial in the numerous cases which result from weakness of the abdominal muscles.

Massage has sometimes been recommended with the object of directly forcing fæces along the colon. Not only do x-ray observations show that this can rarely be done, but, if much force is used, there is considerable danger of injuring the intestinal mucous membrane in the attempt. It is, however, possible to soften fæcal masses in the descending and iliac colon by pressure, so that their subsequent removal by enemata is greatly facilitated.

Spastic constipation is likely to be aggravated by massage unless the rubbing is extremely gentle; ordinary massage can, however, be applied in such cases to any part of the colon, such as the cæcum, which is atonic and not abnormally contracted. Massage should never be employed if there is any evidence of inflammatory complications.

The bladder should be emptied immediately before the massage; the patient should be recumbent and his knees raised by a pillow, in order to relax the abdominal muscles as much as possible. The massage should be repeated daily and should be continued regularly for several weeks at least. The best time is before breakfast, as the stomach is then empty, and the massage, being added to the natural morning stimuli to defæcation, may at once result in a normal evacuation. Its duration should at first not exceed ten minutes, but it can be gradually increased up to half an hour. If it causes much fatigue, the time spent in the treatment should be diminished, and it may be necessary to limit it to three times a week. It is also advisable to discontinue the massage during the menstrual periods. Abdominal massage should not cause any pain; the production of pain is an indication that the massage is not being done skilfully, or that the condition is one which should not be treated in this way.

It is important to give definite instructions as to whether direct stimulation of the colon is required, and if so what part is most affected, or whether treatment should be mainly applied to the abdominal muscles. When it is desired to act directly on the bowel, it is usual to begin with massage of the cæcum and pass along the colon, finishing immediately above the pubes,

although it is very doubtful whether there is any real advantage in doing this. A preliminary examination with the x-rays is of great value, as it shows the exact position of the colon and also the part in which the sluggish action is most marked. Whenever possible, I arrange for the masseur to begin his first treatment under the fluorescent screen whilst the bowel is still filled with barium sulphate, so that he can learn the exact position of the colon and the manipulations required to influence it. This is particularly important in cases of ptosis, where the transverse colon has to be drawn out of the pelvis before it

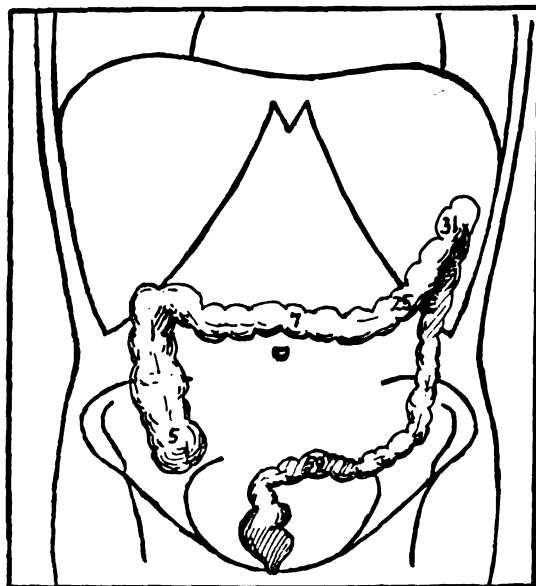


FIG. 1.—Delay in distal part of colon, which is in the normal position. The figures indicate the number of hours after an opaque meal at which the various points were reached.

can be properly massaged. It is remarkable how rarely even the most experienced masseurs are able to recognise the position of the colon without the aid of the x-rays. A comparison between Fig. 1 and Fig. 2 shows how unscientific it is to massage according to set rules without varying the method to suit each individual case.

Only a short description need be given here of the various manipulations, which may be used in the treatment of constipation. The treatment should begin with *stroking*, which consists of slow, light and continuous stroking movements over the abdomen; this is particularly indicated for old, feeble and sensitive individuals, who cannot stand more stimulating

treatment. It is also useful when the abdominal muscles are weak, but it cannot have much effect on the intestines themselves, although the sedative action is a useful preparation for more vigorous manipulations. It is the only form of massage which can be used in spastic constipation without aggravating the condition; sometimes it actually relieves the enterospasm.

*Kneading* is the most useful form of massage in the majority of cases, as it has a powerful action on both the abdominal muscles and the intestines; deep pressure is used and the tissues are kneaded and rolled between the fingers and hands.

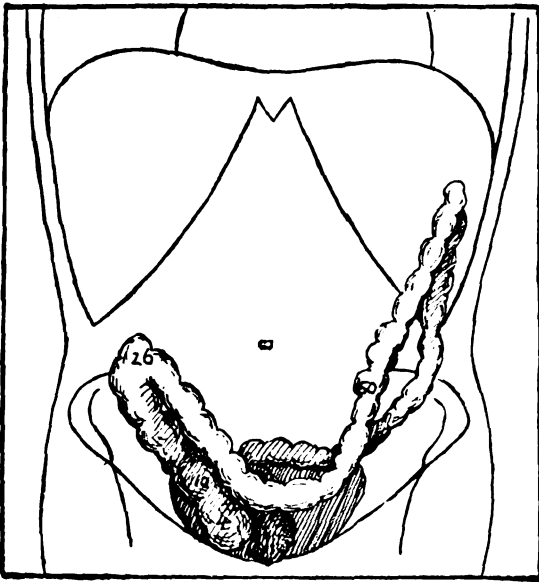


FIG. 2.—Delay in proximal part of a dropped colon.

The efficacy of the treatment is sometimes increased by concluding with *tapotement*, in which the abdomen is sharply struck by a rapid series of strokes with the edge of the hands or the tips of the fingers.

*Vibratory massage* with the aid of more or less elaborate electrical apparatus has been recommended, but the results obtained by its use hardly warrant the trouble and expense involved in carrying out the treatment, as it is not clear that it can do anything which simple massage by the hand cannot accomplish. Moreover, it is possible to combine a certain amount of vibration with the other manipulations, when the massage is done by hand.

*Masso-lavage*.—In 1919 Baumann introduced a combination of intestinal lavage with massage, and the results obtained

both by himself and Reboul at Châtel-Guyon are very encouraging. After a first injection of a pint, which is not retained, a second of a pint and a half or two pints is given. Gentle massage is then performed, beginning with the iliac colon and gradually travelling to the cæcum. By this means the fluid is distributed throughout the colon, and masses of retained fæces are broken up, the bowel being more completely washed out than is possible by any other means. An exceptionally large stool is generally passed from a few minutes to a few hours after the treatment is finished. I have found this combined treatment extremely useful in the early stages of dealing with very severe cases of constipation, in which the whole colon is involved and large quantities of fæces tend to accumulate, especially in the cæcum and ascending colon.

*"Lumbar side-shaking."*—The patient lies on a couch with the knees flexed and feet resting on the couch. The masseur places his hands over the patient's iliac crests and performs a vigorous shaking movement by carrying the hands backwards and forwards, at the same time exerting pressure inwards and downwards. This treatment is particularly useful in the common type of case in which the stasis is most severe in the cæcum and ascending colon.

#### THE LEVATOR ANI MUSCLES IN NORMAL DEFÆCATION AND IN DYSCHIEZIA

In the process of defæcation a peristaltic wave passing down the pelvic colon and rectum directs the fæcal mass towards the coccyx; the normal postural tone of the levator ani muscles directs it towards the entrance of the anal canal, which now opens as a result of active relaxation of the anal sphincters. The final expulsion of the fæces is helped by the voluntary contraction of the levator ani muscles, some of the fibres of which descend in the wall of the bowel and terminate in the fibro-elastic tissue between the external and internal sphincters of the anal canal. Contraction of the levator ani muscles draws the anal canal upwards over the fæcal mass as it is forced downwards by the rectal peristalsis through the relaxed sphincters. Thus both the postural tone and contractile power of the levator ani muscles are important factors in efficient defæcation.

Injury to the levator ani muscles during parturition is a common cause of dyschezia in women, as it leads both to atony and deficient contractile power. By observations made during numerous operations and in a few autopsies, Pincus<sup>9</sup> of Leipzig

found that the levator ani muscles become fragile, pale and relaxed after difficult and prolonged labour, in marked contrast to the other muscles of the body. The change is generally unilateral, and is due to over-stretching associated with anæmia from the long-continued pressure of the foetal head. The postural tone generally returns to normal if the patient remains recumbent for a sufficient period after parturition, as the pelvic floor is subjected to a minimal downward thrust. But in women, especially among the poorer classes, who do not rest long enough, the increase in the postural length and diminution in the postural tone caused by the stretching are perpetuated by the continuous additional pressure of about 15 mm. of mercury on the pelvic floor caused by the weight of the abdominal viscera on the assumption of the upright position and the frequent further strains caused by active movements.

Although difficult labour is the cause of most of the severe cases of dyschezia due to deficient tone and contractile power of the levator ani muscles, a less severe form is very common, either with or without a similar condition of the abdominal muscles, in individuals of both sexes who lead a sedentary life, or who have suffered from any long and debilitating illness.

The anus is normally slightly retracted owing to the tone of the levator ani muscles; the retraction is increased and the anus moves slightly forward when the levator ani muscles are contracted. When the postural tone is deficient, the retraction in the condition of rest is absent or less than normal. Deficient tone is also shown by the effect of straining or making a bearing-down effort, when the whole perineum projects much further than it should do. In severer cases prolapse of the rectum or uterus may be present or may follow straining.

When the contractility of the levator ani muscles is impaired, the normal retraction and forward movement of the anus cannot be produced to the usual extent and it may be entirely absent. The contractile power can be best gauged, however, by direct palpation with the finger in the rectum, when the patient is told to make the same effort she would exert if she wished to overcome an urgent desire to defæcate. If the muscles have been injured, the contraction is found to be weak, particularly on one side.

#### EXERCISES FOR THE LEVATOR ANI MUSCLES

The importance of exercises for the muscles of the pelvic floor has only recently been recognised. Pincus in 1908 appears to have been the earliest writer to recommend them, and in



the first edition of my book on "Constipation," published in the following year, I described how they should be performed. The patient is told to repeat the movement she would do were she trying to restrain commencing defæcation; by this means the levator ani muscles are alternately contracted and relaxed. Following the example of Pincus, I have generally recommended patients to repeat this exercise thirty times every morning and evening whilst in bed. Quite recently Stacey Wilson<sup>10</sup> suggested that the patient should be told to do it many times during every hour of the day, and I am sure that in severe cases this is desirable during the early stages of treatment.

This exercise improves the contractile power of the muscles of the pelvic floor. In most cases their postural tone also requires to be increased. This can only follow more prolonged efforts to keep the muscles contracted. Instead of contracting the muscles and immediately afterwards allowing them to relax, the patient should from time to time during the day contract them and keep them braced up continuously for as long a time as she can, as recommended by Stacey Wilson. This exercise should be performed frequently by women in whom the pelvic floor has been damaged, while they are still in bed, beginning a few days after parturition. By this means the tendency for prolapse of the uterus is likely to be prevented.

These exercises have in an experience of fifteen years proved invaluable in the treatment of large numbers of cases of dyschezia both in men and women. Any tendency to rectal prolapse is at the same time rapidly overcome, and I have seen a number of cases of definite prolapse cured by this means without operation. I was, for instance, consulted in June, 1915, by a lady, who had had an operation performed twelve months before for a very large prolapse of the rectum, but the condition returned almost at once. She had been advised by a distinguished rectal surgeon to have another operation for its relief. Instead of this she succeeded in restoring the normal activity of the muscles of her pelvic floor by exercises. There has been no recurrence in the ten years which have now elapsed, in spite of a very long and serious illness, which resulted for a time in an extreme degree of emaciation.

The same treatment has proved effective for prolapse of the uterus. Some cases of the kind were communicated to me a few years ago by Mr. H. B. Butler of Guildford. In his recent paper on the subject Stacey Wilson confined his attention to the effect of this exercise on cases of uterine prolapse, and did not refer to prolapse of the rectum or dyschezia. He has found that patients were soon able to maintain a previously

extruded uterus in its normal position without the need of a pessary. Curiously enough, however, he regarded the exercise as one for the "vaginal sphincters," but Paramore <sup>6</sup> has pointed out that it is quite clear that it is not the "constrictor cunni" but the levatores ani which are affected by it.

It is obvious from what has already been said about the importance of the pelvic floor in maintaining the normal abdominal pressure, that these exercises will also have the effect of improving the patient's general health. In many cases of visceroptosis and the associated low intra-abdominal pressure they are of even greater importance than the more familiar exercises for the abdominal muscles.

*Sacral Beating.*—"Beating" is a strong form of tapotement; it is performed with the loosely-closed hand and loose wrist. The patient leans forward with his extended legs astride and arms hanging. The masseur stands at his side with the hand supporting his abdomen just above the symphysis pubis. With the other hand he "beats" in small series of five strokes, starting from the middle line and working obliquely outward and downward over the sacrum and gluteal region on each side alternately. This curious form of massage exerts an unexpectedly stimulating action on defæcation, and is frequently of great use in re-educating the rectum to perform its normal function in cases of dyschezia.

I am greatly indebted to Miss W. Warner for her help in describing the details of treatment by exercise and massage. Her exceptional experience in the treatment of abdominal disorders by these methods has made it possible to add practical details to a somewhat theoretical survey of the subject.

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## GENITAL PROLAPSE<sup>1</sup>

By H. B. BUTLER, F.R.C.S.E., Surgeon to Surrey County Hospital, Guildford.

THE subject of genital prolapse is of interest to us all. The condition is a common one and the treatments in vogue are not satisfactory.

Simple rest often gives highly satisfactory results in that class of case where a slight prolapse follows immediately after the puerperal state. When a short term of rest, combined with repair of the perineum, fails to cure, it is usual to fall back on the use of pessaries, in the hope that these may be discontinued after a short period of use. This hope is, however, too often disappointed, and instead of being able to discontinue the pessary after a short time, it may well happen that a larger and more cumbrous instrument has to be worn in order to give the required support, and so on until ventral fixation or suspension of the uterus is undertaken as a last resort. General experience has shown that this operation is by no means always permanently successful in securing the end in view, whichever of the many methods of performing it is chosen.

Since 1913 I have been putting into practice a method of treatment which to the best of my knowledge is new so far as its application to genital prolapse is concerned. I believe it to be rational and based on correct principles, and it has given results far exceeding my expectation, in the comparatively few cases which have come in my way to treat.

It is sometimes stated that a prolapse of any part of the female genitalia is really a hernia, but I would rather define prolapse as "a failure on the part of the pelvic diaphragm to afford sufficient support for the pelvic contents." The condition is really analogous to that of the pendulous abdomen, in which the anterior abdominal wall fails to give sufficient support to the abdominal viscera.

The perineum is a subsidiary support, and it should be properly repaired if it is damaged. It is, however, conceded on all sides that the integrity of the perineum has a comparatively small influence on the production, prevention or cure of prolapse, and though the repair of the perineum, with an extensive

<sup>1</sup> A preliminary report was read before the Guildford Division of the British Medical Association, February 27, 1914.

anterior and posterior colporrhaphy, may make the opening so small that the genitalia are unable to come down, the real defendant—the pelvic diaphragm—is only supported from below by a mass of scar tissue, which often stretches in time, with the reproduction of the original condition.

### *Anatomy*

The pelvic diaphragm consists of pelvic fascia above, next a flat sheet of muscle and below this the anal fascia.

The most important of these is the voluntary muscle, which is attached in front to the back of the symphysis pubis, at the sides to the white line and the spine of the ischium, behind to the sacrum. The anterior part of this muscle is known as the levator ani, the posterior part as the coccygeus, but for practical purposes the whole sheet may be regarded as one muscle. This muscle passes downwards and inwards towards the middle line, where it meets its fellow of the opposite side and with it forms a diaphragm which, with the pelvic fascia overlaying it, shuts out the perineum from the abdominal cavity. This diaphragm is perforated in the midline by the rectum behind, and by the uterus, vagina and urethra in front. These structures do not pass through a mere decussation in the muscle, as the aorta does through the thoracic diaphragm, but the muscle is actually inserted into the penetrating viscera by muscle fibres sent down along their walls. It may therefore be said, using the ordinary anatomical meaning of the words, that the muscle arises from the fixed points, the bony attachments and the white lines, and is inserted into the penetrating viscera.

It follows that freedom from prolapse absolutely depends on the integrity of this muscular sheet, which when functioning properly makes prolapse an impossibility.

There are some points of similarity between the two diaphragms—pelvic and thoracic, the one at the lower and the other at the upper end of the abdominal cavity. The dome of each is increased when the muscle is relaxed and diminished or flattened when contracted. The concavities of the domes being opposite to one another, the thoracic diaphragm when inefficient is raised and the pelvic diaphragm when inefficient is lowered.

### *Pathogenesis*

The acute and most frequent cause of genital prolapse is the pressure, stretching and bruising of the pelvic diaphragm during childbirth. Bruising of the nerve supply is probably an additional factor in the delayed recovery of the muscles concerned.

The violent and futile efforts to expel the fæces in dyschezia may further increase the strain on the already relaxed and stretched muscle fibres.

Atoxic neuritis of the pelvic nerves caused by chronic septic absorption from infected pelvic organs may also aggravate the condition. It must also be remembered that if the pelvic diaphragm is out of action from any other cause its fascia may become a site for sub-infection from some primary focus not necessarily in the pelvis—such as teeth or tonsils. I suggest that the painful condition which results is of a “rheumatic” or fibrositic type, and analogous with those conditions which give rise to flat foot in gonorrhœal rheumatism. I believe that some of the cases of chronic pelvic pain, associated with retroversion of the uterus and some degree of prolapse, may be of this origin.

Chronic over-fatigue of the muscle in supporting the weight of a fibroid uterus or other pelvic tumour may also lead to prolapse. When degenerative changes occur in these tumours, the toxic factor may again come into play.

These, as far as I know, are the principal causes in the production of genital prolapse, and I believe that such causes as laborious occupations, long standing, chronic cough, are rarely capable of producing prolapse, in the absence of one or other of the above-mentioned factors, though they may be very effective means of aggravating the condition. The first essential must then be to determine the cause and to treat that efficiently.

### *Treatment*

In toxæmic cases, the primary focus should be sought for and treated. If a prolapsed uterus is itself diseased and has to be removed, it must be remembered that its removal will not cure the prolapse of other viscera. Lastly, a fibroid uterus or any other tumour, which is causing a prolapse, must be removed. It sometimes happens that a large fibroid uterus, which has almost filled the pelvis and bulged over the brim and is far too large to become prolapsed itself or to allow other viscera to do so, is removed and prolapse promptly follows, because the levator ani and its nerves, long stretched and pressed between bone and tumour, is quite unable to support the weight of the viscera which now descend and occupy the pelvis. All these conditions are, I believe, suitable for treatment by the means I shall describe.

Whatever the cause of prolapse may be and however that

cause be relieved, prolapse will continue, unless the efficiency of the levator ani muscle is re-established. To borrow an illustration from Crile. One might as well advise an impending bankrupt to relieve his condition by increasing his expenditure, as expect to regenerate an over-loaded and over-stretched muscle by increasing the stretch and increasing the load. Such is the treatment by pessaries. Suspending the uterus from above does not attain the desired end, for when it fails, as it often does, prolapse recurs, showing that the muscle has not taken advantage of its temporary rest to regenerate itself.

Supporting the muscle from below, by means of perineorrhaphy and colporrhaphy operations, also sometimes fails, and after a time the pelvic viscera again come down.

This shows that in chronic cases, where the muscle has been out of action for a long period, recovery of tone does not take place as the result of rest alone. It follows, therefore, that, even if these operations are done, some other treatment should also be given to re-establish the health and vitality of the pelvic diaphragm.

The muscle of the pelvic diaphragm is a voluntary muscle; it can therefore be exercised at will, in the same way as any other voluntary muscle in the body and with the same results. Voluntary exercise is absolutely necessary to any damaged limb before it can be restored to proper function, and so it is to the levator ani or any other muscle which has been put out of action for a prolonged period. It is obviously necessary that a muscle which is weak and over-tired should be relieved from the continual strain of supporting its load—either entirely or at intervals according to the severity of the case.

This object may be attained by putting the patient to bed, with the foot end raised on blocks of about ten inches.

A patient who is not sufficiently crippled by her condition to be completely bedridden should nevertheless remain in bed until after breakfast, should return to rest in bed for an hour and a half in the afternoon and go to bed for the night at nine o'clock. During all these periods of rest the strain on the pelvic diaphragm is entirely relieved.

Three times a day—before rising in the morning, at the end of the afternoon rest period, and an hour after going to bed at night—the exercises should be performed.

I may mention that these levator ani exercises, in addition to strengthening the muscle and enabling it to support the pelvic contents, are of great value in the treatment of any dyschezia.

The exercise consists of making alternate strong contractions

and relaxations of the levator ani, coccygeus, and accelerator urinæ muscles, acting together.

The strength of these contractions may be increased by relaxing the thoracic diaphragm, drawing in the muscles of the abdominal wall and pulling up the abdominal viscera.

It is often difficult to explain to a patient in what manner a contraction of these muscles may be made, but I have found the following illustrations useful. With regard to the main portion of the muscle, the movement of drawing in the rectum, which she would use to retain an enema injection that she urgently desired to release. With regard to the anterior part, the movement which would be made if she wished to stop passing water in the midst of the act of micturition.

I generally suggest that these exercises should be performed twenty times, that is to say, twenty contractions and twenty relaxations, at the end of the rest periods and shortly after retiring to bed for the night.

In conclusion I will narrate three cases to illustrate the effect of this treatment.

(1) A patient, æt. 30, after the birth of her second child, suffered from prolapse as soon as she got up. She returned to bed for a fortnight, but on getting up again the prolapse returned, and on straining to defæcate the cervix presented at the vulva. She was given a pessary, which she continued to wear for nine months.

At the end of that period the pessary was removed altogether, but the prolapsed condition was in no way improved. She was then treated in the way described. After a fortnight she began to be aware of improvement and was more comfortable than she had been even while wearing the pessary, and at the end of three months she was completely cured.

(2) A patient, æt. 64, had a fibroid uterus about the size of a large orange. She had a great deal of pain, with prolapse resulting in the appearance of the cervix at the vulva and a cystocele, which also gave her a great deal of pain and inconvenience.

On examination the uterus proved to be tender and the fibroid was judged to be degenerating; she was also tender in the region of the appendix.

Abdominal hysterectomy was performed and the appendix, which was also inflamed, was removed at the same time. An attempt was made to sling the stump of the uterus by sewing this to the remains of the round ligaments.

When this patient got up the prolapse immediately became troublesome and the cystocele especially gave rise to a great deal of trouble and annoyance. She was given a pessary, with which she got about in comparative comfort for a year.

At the end of that period the pessary was removed altogether, but the prolapse and cystocele were as bad as ever, and the patient declared that it would be impossible for her to walk or get about at all unless she had some mechanical support.

She was put on to the exercises and rest treatment; no pessary or other support was again used. She persevered, and at the end of a month had made such good progress that she declared herself better than ever before. At the end of six months there was hardly any descent of the uterine stump even on straining and the cystocele has almost entirely disappeared. In the twelve years which have since elapsed she has lived an active life and has never again worn a pessary.

(3) A patient, æt. 29, had a slight prolapse and chronic retroversion after her first baby. She wore a Hodge pessary to relieve this condition for six months, and until the second child was four months *in utero*. After its birth the uterus again became retroverted, and when I first saw her, ten weeks after the baby was born, she was in a condition of chronic pain. She had a large, soft, subinvolted uterus, retroverted and tender. She also had severe flooding at her period. She was too tender to bear any pessary.

After a week of ichthyol tamponage, she was given a gelatine padded ring pessary, size 12 (the smallest that would remain in).

The uterus gradually got smaller, but the pain persisted and the uterus obstinately returned to the retroverted position, with increased pain, whenever the pessary was removed. Leucorrhœa was persistent and periods excessive.

At this stage, six months after the birth of the baby, the ring was removed altogether, and the patient was put on the treatment described. The pain gradually lessened and the uterus resumed an upright position. Four months after the removal of the pessary, not only had all pain ceased, but the uterus, which has become firm and healthy, no longer descended and had become anteverted. The leucorrhœa had ceased, the periods were normal and all the pelvic organs were in a healthy condition.



## SOME OBSERVATIONS ON THE CUTANEOUS TUBERCULIN TEST OF VON PIRQUET

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THE work described here was begun with the object of finding whether the reaction in clinically non-tuberculous persons, who give a positive result with the test, could be differentiated from that given by tuberculous patients. For this purpose it was thought necessary to use a technique which involved the treatment of the same extent of skin surface with the tuberculin in every case, so that any variations in the size of the resulting papule could be measured and compared. It was observed, however, that the two types of cases could not be distinguished merely by comparing the degree of reaction.

On comparing the measurements, which in the majority of the cases were made for seven consecutive days, it was noticed that certain differences could be made out in the way the reaction developed in the two types of cases. In clinically non-tuberculous persons the papule usually goes on increasing in size after the first twenty-four hours, reaching the maximum on any of the subsequent days, after which it begins to decline. Patients suffering from tuberculosis behave in two different ways: (1) the greater number give a reaction which is maximal on its first appearance; (2) a smaller proportion respond in a manner similar to that met with in non-tuberculous persons. These variations in tuberculous patients can, in a large number of cases, be correlated with different clinical phases of the disease.

Tuberculous patients suffering from extensive or acute disease, some of whom have since died, gave a positive result of a nature suggesting poor reactivity. The papule in such cases is generally small and slightly raised, and tends to become discoloured or to disappear completely towards the latter half of the period of observation.

### THE TECHNIQUE

The technique employed is a modification of that used by McNeil.<sup>1</sup> Two circular areas are marked out on the flexor aspect of the forearm below the elbow-joint by pressure with

the cap of a clinical thermometer case. The areas are 7 mm. in diameter. The skin is cleansed with ether and chafed by means of a sharp darning or carpet needle so as to remove the epidermis all over these areas, care being taken to avoid bleeding.

Human tuberculin 100 per cent. (Koch's old tuberculin) is placed on one area by means of a sterile 4 mm. loop of platinum wire, and the skin is further scratched with a Von Pirquet scarifier, holding the instrument on the slant so as to use its edge rather than the point. The other area is used as the control. A loopful of 50 per cent. glycerin in saline solution, to which 0.5 per cent. phenol is added, is placed on this spot, which is scarified as described above.

The spots are allowed to dry; the part is then covered with a piece of lint which is held in place by plaster for twenty-four hours. Observations are made at the end of this period and are repeated daily for a week. A positive reaction is indicated by the appearance of a more or less raised papule. A pink spot which is not raised is not regarded as a positive reaction, since it is usually replaced by a papule, and in one instance the latter appeared after the spot had become discoloured. The papule is measured with a small pair of calipers and a millimetre scale in two directions at right angles to each other, and the same diameters are measured on successive days. If the papule is quadrilateral in outline the distances between the opposite sides are measured. The margin of the papule is frequently ill-defined, but with a little care it can be made out. The elevation of the papule is also noted. If the papule becomes discoloured the fact is recorded.

A result is not considered to be negative until the end of a week, as the appearance of the papule is sometimes delayed up to the second or third day. In two of my cases it appeared as late as the fifth day. McNeil <sup>2</sup> has noted delayed reactions in children.

#### RESULTS AND DISCUSSION

The local reaction varied from a papule 2 or 3 mm. in diameter to one 35 mm. across. It may be slightly raised or stand out prominently. When the local reaction is very well marked, a red flush often forms an areola around the papule, or vesicles appear on the surface of the papule. In five cases there was a red streak of lymphangitis along the inner aspect of the arm, and pain in the axillary glands, but these subsided in two or three days without producing a constitutional reaction.

There was no general reaction, though in pyrexial cases it is

difficult to judge this. In one patient headache and pain in the neck and corresponding shoulder were complained of forty-eight hours after the scarification, and the temperature rose to 99° F., but she was subject to similar rises of temperature once or twice a week.

The reaction which reaches its maximum size on the day of its first appearance is usually seen in tuberculous cases with toxæmic symptoms, and is for convenience called the type "A" reaction. Chronic cases and patients who have made a good response to sanatorium treatment generally give a reaction which attains the maximum size one or more days after its first appearance. This type of reaction is also given by the great majority of control cases and is spoken of here as the type "B."

In three of the control cases and two of the tuberculous patients the papule remained of the same size from the first to the seventh day.

The test was performed on 110 cases. Of these 52 were

TABLE I.  
ANALYSIS OF CONTROL CASES.

Total number of cases.	Negative result.	Type "B" reaction.	Type "A" reaction.	Reaction did not change in size.
52	10 = 19·2%	37 = 71·2%	2 = 3·8%	3 = 5·8%

controls suffering from various non-tuberculous diseases, and 58 were patients suffering from tuberculosis, the majority being pulmonary cases.

The results in the 52 control cases are analysed in Table I. Details of the reaction are shown in Table II.

Of the 52 control cases 42, *i.e.* 80·8 per cent., gave a positive reaction; and 10, *i.e.* 19·2 per cent., were negative. Of the 42 positive cases 37, *i.e.* 88·1 per cent., gave the type "B" reaction; in 2, *i.e.* 4·8 per cent., the reaction was of type "A"; while the remaining 3 cases showed no alteration in the size of the papule in the course of seven days.

Neither of the two cases which showed the type "A" reaction had any sign of tuberculous disease clinically. Case No. 6 (Table II) was under treatment for diabetes mellitus, and case No. 12 had auricular fibrillation, probably following upon influenza. These cases were encountered early in the course of the work when the papules were measured along only one

diameter. Later the possibility of enlargement of the papule in other directions than that measured was realised, and measurements were made along two diameters. In one of the later cases such an error was avoided by following this method, and, perhaps, was incurred in these two cases.

TABLE II.  
MEASUREMENTS IN CONTROL CASES.

Case No.	Age in years.	Disease.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
			Negative						
5	11	Chorea.							
6	42	Diabetes mellitus with peripheral neuritis and acidosis.	7	7	7	6	6	6	6
7	46	Ulcerative colitis.	2	3	3	3	3	3	Faded
8	32	Cholecystitis.	5	7	8	10	9	9	9
9	53	Diverticulitis.	10	10	12	11	11	11	11
10	34	Disseminated sclerosis.	6	6	6	8	7	7	7
11	59	Auricular fibrillation.	7	7	7	7	7	7	7
12	17	Auricular fibrillation.	0	7	6	5	5	5	5
14	38	Duodenal ulcer.	6	11	12	12	15	15	15
15	23	Chlorosis.	9	13	14	15	16	15	12
16	23	Rheumatoid arthritis.	9	10	10	12	11	11	11
17	28	Asthma.	9	9	9	9	9	9	9
18	30	Gastric ulcer.	13	20	20	25	25	25	23
19	16	Rheumatoid arthritis.	15	15	18	16	16	16	15
20	30	Gastric ulcer.	5	8	10	10	10	10	10
21	18	Rheumatoid arthritis.	6	6	6	6	6	7	9
22	55	Cerebral thrombosis.	16	18	17	19	20	20	20
23	61	Pernicious anaemia.	6.5	6.5	11	11	12.5	12	12
24	33	Chlorosis.	0	9	11	10	12	12	13
25	13	Diabetes mellitus.	8	9	9.5	11.5	11	12	12
26	59	Sciatica.	9	13	12	11	11	11	11
32	30	Colitis (origin not known).	0	6	10	10	7	7	6
33	14	Steatorrhoea.	8	8	10	10	9	7.5	7
35	29	Dyschezia.	12.5	16	23	25	25	24	22
36	36	Bronchiectasis.	12	16	17	14	14	14	14
37	40	Peritoneal adhesions.	9	11	12	12			Patient was discharged
38	66	Emphysema of lungs.	13	15	16	15	15	14	14
39	34	Gastric ulcer.	14	16	15	15	14	13.5	13
40	40	Duodenal ulcer.	10.5	11.5	13	10.5	11	13	13
42	41	Fibroid lung (non-tubercular).	8.5	9	10	10	10	9	9
46	22	Appendicitis (chronic).	Negative.						
48	46	Diabetes mellitus.	16.5	17	20	20	20	20	20
49	40	Duodenal ulcer.	16	20	25	25	24	22	19
50	58	Auricular fibrillation.	Negative						
51	42	Duodenal ulcer.	0	0	0	0	7	8	8
52	59	Pernicious anaemia.	23	24	24	24	24		Patient was discharged
53	39	Gastric ulcer.	15	17	18	18	18	18	18
54	25	Gastric ulcer.	Negative						
55	58	Cystitis.	Negative						
56	50	Diabetes mellitus.	0	6	9	8	7	7	7
57	35	Gastric ulcer.	0	8	12	11	11	11	10
61	17	Sinus leading to ischium and astragalus resulting from abscesses following lobar pneumonia.	Negative						
86	51	Rheumatoid arthritis.	0	2	2	3	3	3	3.5
87	13	Rheumatic arthritis and mitral stenosis.	Negative						
88	35	Gastric ulcer.	22	35	17	17	15.5	15	14
89	41	Diabetes mellitus.	10	11	13	13	14	14	14
90	34	Chronic nephritis.	7.5	20	19	17	16.5	16.5	16
91	17	Diabetes mellitus.	Negative						
95	42	Abscess of the lung.	11.5	15.5	16.5	15.5	13.5	13.5	13.5
96	13	Skin eruption which was regarded as ? Lichen scrofulosorum, but soon cleared up.	Negative						
97	24	Hodgkin's disease.	Negative						
98	24	Enlarged glands in neck (syphilitic).	0	0	0	0	11	13	13

The results of the 58 tuberculous cases are analysed in Table III.

TABLE III.  
ANALYSIS OF TUBERCULOUS CASES.

Total number of cases.	Negative result.	Type "A" reaction.	Type "B" reaction.	Reaction did not change in size.
58	0	38 = 65.5%	18 = 31.0%	2 = 3.5%

This series, in which are included ten cases with a grave prognosis, gave a positive reaction in every case: 38 cases, *i.e.* 65.5 per cent., showed the type "A" reaction; 18, *i.e.* 31.0 per cent., gave the type "B"; and in 2, *i.e.* 3.5 per cent., the papule did not alter in size in the course of seven days.

The measurements of the papules in the 38 tuberculous cases which gave the type "A" reaction are shown in Table IV.

TABLE IV.  
TUBERCULOUS CASES WHICH GAVE THE TYPE "A" REACTION.

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
		7	7	6	6	6	5	5
1	A. A. Male, <i>et.</i> 24. Phthisis right apex and extensive disease shown by x-ray in left lung also. Febrile. Loss of weight. Sputum positive. Died two months later.	7	7	6	6	6	5	5
2	V. G. Female, <i>et.</i> 21. Enlarged glands right side of neck and pleurisy with effusion right side. Pyrexia, night sweats and loss of weight. Sputum negative.	16	16	13	13	13	12	12
3	H. S. Female, <i>et.</i> 22. Phthisis left apex and extensive disease shown by x-ray in left lung. Has pyrexia and loss of weight. Sputum positive.	14	14	13	9	4	4	4
13	O. J. Male, <i>et.</i> 39. Phthisis apices of both lungs, pleural effusion right side, tuberculous epididymitis both sides (confirmed by microscope after operation) and orchitis on left side. Pyrexia and loss of weight. Sputum negative.	12	12	12	12	12	12	11
28	E. L. Female, <i>et.</i> 20. Phthisis left lung (extensive disease) since January 1923. Kept well until May 1924, after this she began to lose weight but remained afebrile and artificial pneumothorax had to be done on the left side. Sputum positive.	11	11	11	9.5	9.5	9.5	9.5
29	E. H. Female, <i>et.</i> 17. Phthisis. Extensive disease both lungs. Pyrexia and loss of weight. Sputum positive. Sent home as a hopeless case.	0	9	9	9	7.5	5.5	5.5
30	F. S. Male, <i>et.</i> 18. Phthisis left apex (early case). Evening rise of temperature. Pulse rate 100. Sputum not available.	20	20	19	17	17	15	15
31	A. F. Male, <i>et.</i> 27. Phthisis. Fibroid condition left lung. Pyrexia and loss of weight. Thoracoplasty done on left side. Sputum positive.	18	14	13	13	12	12	12

TABLE IV (continued).

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
		16	14	12	12	12	12	12
34	E. C. Female, <i>et.</i> 30. Phthisis right lung. Was treated by artificial pneumothorax but developed pyrexia and night sweats and lost weight. Thoracoplasty was performed. Sputum positive.	16	14	12	12	12	12	12
41	C. H. Female, <i>et.</i> 24. Phthisis right apex since 1920. In January 1924 returned home after a second course of sanatorium treatment, but began to lose weight and had pyrexia, and artificial pneumothorax was commenced. At the time the test was performed she was still showing a slight rise of temperature. Sputum positive.	21	21	20	19	19	19	18.5
44	J. B. Male, <i>et.</i> 23. Phthisis right apex. Early case. Had hemoptysis three weeks ago. Has pyrexia and slight wasting. Confirmed by x-ray. Sputum not available.	35	35	35	32	32	31	31
62	E. B. Male, <i>et.</i> 35. Phthisis right apex. Hemoptysis in 1914 and another in 1924. No pyrexia. Weight steady since 1915, and put on weight while in sanatorium. Walks four miles a day. No sputum. Was discharged from sanatorium as fit for work just after the test.	23	23	16	15	15	13	13
63	E. R. Male, <i>et.</i> 15. Pott's disease with sinus in back of right thigh. No pyrexia. Weight cannot be taken as patient is confined to bed. Pulse rate in evening 72 to 104.	11	11	11	11	10	10	10
64	A. H. Male, <i>et.</i> 9. Old tuberculosis of lung, spine and right hip-joint; and subsequently had multiple subcutaneous abscesses which have healed. Has a slight rise of temperature two or three days a week.	12	12	12	11	11	10	10
66	A. McL. Male, <i>et.</i> 17. Phthisis both apices. Has pyrexia and loss of weight. Sputum positive.	12	12	12	11	10	10	10
67	G. E. Male, <i>et.</i> 38. Phthisis both apices. Is febrile at times, and has had night sweats recently. Is gaining weight at sanatorium but is still below weight in health. Pulse rate 80 to 100. Sputum positive.	10	10	10	10	10	9.5	9.5
68	R. B. Male, <i>et.</i> 55. Phthisis both apices. Slight evening pyrexia on some days. Has lost weight but is regaining it at sanatorium. Sputum positive.	13	12	11	11	11	10.5	9.5
69	E. W. Male, <i>et.</i> 40. Phthisis both apices. Has an evening rise of temperature. Has lost weight but is regaining it at sanatorium. Sputum positive. Later reported to be again losing weight.	28	28	28	28	28	24	24
72	E. V. Male, <i>et.</i> 42. Phthisis both lungs. Pott's disease with abscess in right loin, and right tuberculous orchitis and epididymitis. Pulse rate 84 to 92. Has evening pyrexia, which has increased since the test. Sputum positive. Weight cannot be measured as patient is confined to bed.	9	8	8	8	8	8	8
75	G. R. Male, <i>et.</i> 33. Phthisis right apex and tuberculous laryngitis. Has a slight rise of temperature occasionally. Weight rising and falling slightly. Sputum positive. Two months later he had daily rise of temperature and was losing weight.	16.5	16.5	14.5	14.5	12.5	11.5	11.5
76	F. H. Male, <i>et.</i> 39. Phthisis left apex and tuberculous laryngitis. Is febrile and is losing weight. Sputum positive.	11.5	11.5	11.5	11.5	11.5	10	8.5
78	S. T. Female, <i>et.</i> 45. Pott's disease with lumbar abscess on right side. Weight cannot be measured as patient is confined to bed. Afebrile. Pulse rate 88 to 96 in bed. Had pleurisy six weeks later.	14.5	14.5	14	12	12	12	12

TABLE IV (continued).

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
79	M. T. Female, æt. 21. Phthisis left apex (early case). Febrile. Is regaining weight. Sputum positive.	15	14	13	12.5	12	12	12
81	E. D. Female, æt. 17. Phthisis left apex. Has an occasional rise of temperature. Is regaining weight. Pulse rate 80 to 100. Sputum positive.	13.5	13	13	13	12	12	12
82	D. B. Female, æt. 23. Phthisis right apex. Has an occasional rise of temperature. Evening pulse rate 80 to 92. Is gaining weight in sanatorium. Sputum positive.	10.5	10.5	9.5	9.5	9	9	8.5
83	I. S. Female, æt. 17. Phthisis right base. Slight rise of temperature once or twice a week. Evening pulse rate 96 to 128. Slight gain in weight. Sputum positive.	19	14	12	12	12	12	12
84	H. B. Male, æt. 31. Tuberculous glands in neck. Has frequent rises of temperature.	0	0	9.5	7	6	6	5.5
85	M. R. Female, æt. 17. Acute pneumonic tuberculosis of both lungs. Febrile case with rapid wasting. Sputum positive.	4.5	4	3	Reaction completely discoloured			
92	W. H. Male, æt. 41. Phthisis. Both apices show suspicious signs. Slight evening rise of temperature. Sputum negative. X-ray showed evenly distributed opacity in right lung, and some infiltration in left lung. This case came immediately after an attack of hæmoptysis.	17	15.5	15.5	14	13.5	13	12
93	E. D. Male, æt. 44. Phthisis left apex and pleurisy with effusion left base. Febrile. Sputum positive.	13	12	10.5	9.5	9.5	9.5	9.5
94	N. R. H. Female, æt. 27. Phthisis. Extensive disease both lungs and tuberculous enteritis. Pyrexia, night sweats and loss of weight. Sputum and faeces positive.	14	13	13	12	11	10	9
99	N. F. Female, æt. 21. Bazin's disease and papulo-necrotic tuberculide. Has tuberculous glands above the left clavicle and in the left axilla. Has no pyrexia and there is no loss of weight. Bazin's disease is spreading.	18.5	18	16.5	16	(Measured for four days only)		
102	A. W. Male, æt. 49. Phthisis both apices and tuberculous laryngitis. Has occasional evening rise of temperature. Has lost weight. Sputum positive. Later reported to be gaining weight.	18.5	18.5	17.5	16.5	15.5	13.5	
103	W. N. Male, æt. 29. Phthisis. Extensive disease right lung. Febrile. Weight rising and falling. Sputum positive. X-ray shows mottling in left lung also. Later report says weight is still rising and falling.	8	7.5	7.5	7.5	7.5	7.5	
105	F. N. Male, æt. 42. Phthisis right apex. Febrile case. Rather anæmic. Is losing weight. Sputum positive. Later report says he is still losing weight.	12	11.5	11	9.5	9.5	9	
106	F. S. Male, æt. 34. Phthisis right apex and base. X-ray shows disease in left apex also. Afebrile since admission to sanatorium but had an evening rise of 99.6 F. after walking two miles. Sputum positive. Had pleurisy three months ago.	21	17.5	16	16	15.5	15.5	
108	E. P. Female, æt. 38. Phthisis both apices and tuberculous laryngitis. Has a slight rise of temperature now and then though confined to bed. She thinks she has lost weight recently. Sputum positive. Later reported to be progressively losing weight.	13.5	12.5	Reaction became completely discoloured.				
109	E. K. Female, æt. 37. Phthisis. Signs in left apex and right axilla. Has an occasional rise of temperature though confined to bed. Weight is on the rise. Sputum positive.	26	24	21.5	20.5	19	19	

Of the 38 tuberculous cases which gave the type "A" reaction, 36, *i.e.* 94.7 per cent., gave evidence of the presence of tuberculous toxæmia in greater or lesser degree, though in two of these patients (Nos. 63 and 78) an evening rise in the pulse rate was the only sign of toxæmia obtainable. Both of them had Pott's disease and had been in residence at the sanatorium for some time, about ten and five months respectively. From the nature of the disease they were confined to bed, and their weight and constitutional reaction to graduated exercise could not be judged. The remaining two cases showed no signs of toxæmia (Nos. 99 and 62). The former patient had Bazin's disease and papulo-necrotic tuberculide, complicating an older tuberculous lesion of the glands in the neck and axilla. She had no signs of toxæmia, and the patch of Bazin's disease had spread recently. Patient No. 62 had an attack of hæmoptysis in 1914 and another in 1924. The physical signs at the apex of the right lung were impaired resonance, granular breath sounds and crepitations; and x-ray examination showed mottling in this region. He had no sputum, no signs of toxæmia, and no evidence of a tuberculous complication. His condition was so good that he was discharged to return to his work.

It is not uncommon to come across patients in whom the presence of tubercle bacilli cannot be demonstrated and the clinical examination does not yield sufficient evidence for a definite diagnosis of tuberculous disease. In such cases, if the patient gives the type "A" reaction, it is a useful aid in diagnosis.

The measurements in the 18 cases which gave the type "B" reaction are shown in Table V. Four of these (Nos. 27, 43, 65 and 77) were chronic cases with arrested disease, seven (Nos. 58, 60, 71, 73, 80, 100 and 101) were patients who had responded well to sanatorium treatment and, except case No. 80, a female, were employed four hours a day in the workshop. Three more (Nos. 104, 107 and 110) had not yet become completely afebrile, but showed much improvement under sanatorium treatment. Thus out of the 18 cases which gave the type "B" reaction, 14 were patients who had improved under treatment, and in 11 of these, *i.e.* 61.1 per cent., the improvement was quite definite. This result is interesting, since it is generally agreed that the majority of clinically non-tuberculous persons, who also give the type "B" reaction, harbour an old arrested tuberculous lesion.

Of the remaining four cases, one (No. 45) was an old case of dry pleurisy; the patient had recently become run down, and



TABLE V.

TUBERCULOUS CASES WHICH GAVE THE TYPE "B" REACTION.

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
		8	9.5	11	11	10.5	9	9
27	E. T. Male, aet. 12. Tuberculous glands in the neck. He was treated for this from 1919 to 1921 and has kept well since. He is afebrile and comes up for observation only.	8	9.5	11	11	10.5	9	9
43	E. M. Female, aet. 25. Chronic phthisis right apex. Is under observation and treatment since 1920. Gets run down after pregnancy and has been twice in sanatorium (1920 and 1924). Afebrile. Sputum positive.	17	19	19	19	19	19	19
45	G. G. Male, aet. 22. ? Phthisis. Had dry pleurisy three years ago on right side. Three weeks ago had cough and pain on left side of chest. Has lost weight and appetite recently and has pyrexia. Impaired resonance and harsh breath sounds at right apex. X-ray shows diffuse opacity at right apex. Sputum negative. Condition was detected early and he improved rapidly at the sanatorium.	0	7.5	9	10	11	14	14
58	A. T. Male, aet. 25. Phthisis both apices. Has responded to treatment at sanatorium, where he works four hours a day. Afebrile. Sputum positive.	11	14	14	14	13.5	12.5	12.5
60	R. L. Male, aet. 40. Phthisis left apex. Has responded to treatment at sanatorium, where he works four hours a day. Afebrile. Sputum positive.	10.5	12	12	12.5	12.5	8.5	8.5
65	I. K. Female, aet. 36. Pleurisy with slight effusion two years ago. Well since.	10	14	14	13.5	12	11.5	11.5
71	F. L. Male, aet. 15. Phthisis right apex since 1922. Has responded to treatment at sanatorium, where he works four hours a day. Afebrile. Sputum positive.	14	14	14	14	14.5	14.5	13
73	W. B. Male, aet. 25. Phthisis left apex. Has responded to treatment at sanatorium, where he works four hours a day. Afebrile. Sputum negative.	8	9	10	10.5	11	10.5	9.5
77	C. G. Male, aet. 47. Lupus on left hand since 1918. Afebrile. Is under treatment with arc-lamp.	18	20	20	20	17	17	16
80	L. Y. Female, aet. 18. Phthisis right apex. Detected early owing to haemoptysis and treated early. Afebrile from onset. General condition good. Sputum negative. Diagnosis confirmed by x-ray. Is allowed to be out of bed six hours a day and move about in the ward.	11	13	13	12	10	9.5	9.5
100	J. C. Male, aet. 39. Phthisis both apices. Has responded to treatment at sanatorium, where he works four hours a day. Is gaining weight and is afebrile. Sputum negative.	9	10	10	11	12	11.5	
101	G. B. Male, aet. 34. Phthisis both apices since 1923. Readmitted to sanatorium early in 1925 as he began to have a slight rise of temperature. Is afebrile at present and works twenty hours a week. Sputum positive.	22	25	20.5	20	20	20	
104	L. P. Male, aet. 27. Phthisis right apex. Has a slight rise of temperature very occasionally. Walks two miles a day. Sputum negative.	10	11.5	12	12.5	12.5	11.5	
107	E. P. Female, aet. 30. Phthisis left apex. Detected in September 1924, and treated at sanatorium, but still gets a slight rise of temperature two or three times a week. Sputum positive.	11	16.5	15.5	13.5	12.5	12.5	

TABLE V (continued).

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).						
		11	13.5	12	11	11	11	
110	W. B. Female, aet. 14. Phthisis right apex. Detected early and treated early. Has been at sanatorium for five weeks. Is still febrile, but has gained 5 lbs. 11 oz. in weight. Sputum positive.							
70	F. A. Male, aet. 25. Phthisis both apices. Has neither improved nor got worse at the sanatorium, being still febrile and has not put on weight. Sputum positive.	12	12	15	14	14	14	14
41	These cases are described in Table VI.	6	8	8	6	6	6	6
59		5	8	8	Reaction became completely discoloured			

developed cough and a pleural rub with pyrexia and loss of weight. The condition was detected three weeks after the onset, and he responded promptly to sanatorium treatment. Two others (Nos. 4 and 59) were cases with a grave prognosis. They will be described below with others of a similar nature. The last case (No. 70) cannot be explained. He had remained toxæmic in spite of sanatorium treatment, and clinically appeared to have neither improved nor got worse.

These results show that a large proportion (94.7 per cent.) of the patients who give the type "A" reaction are clinically cases in which the toxæmia and local lesion are not well under control, while among cases which show the type "B" reaction, 61.1 per cent. show definite improvement. But the presence or absence of toxæmia and activity of the local lesion cannot be the full explanation for two reasons. Among patients who give the type "B" reaction there are some who show signs of toxæmia, while patient No. 62 described above, who had no toxæmia and was clinically fit to resume his occupation, gave the type "A" reaction. Therefore there are other probable factors, not evident in the clinical examination of the patient, which determine the type of reaction. What Wingfield<sup>3</sup> calls the "margin" or "balance of resistance" to the credit of the patient may possibly have something to do with it. In this respect it would be interesting to note the reaction in patients at the time they are discharged to resume their work, and to trace their subsequent history.

The character of the reaction in the following six cases presents points of interest. They are cases of extensive or acute disease (Table VI).

Three of these cases (Nos. 1, 29 and 85) gave the type "A" reaction; in two (Nos. 4 and 59), contrary to expectation, it

TABLE VI.

CASES WITH A GRAVE PROGNOSIS.

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).
1	A. A. Male, <i>act.</i> 24. Had been ill seven weeks. Had signs of phthisis at right apex. X-ray showed affection of the right apex and extensive shadows in the left lung. Pyrexia and loss of weight. Died at home two months later of acute miliary tuberculosis.	7   7   6   6   6   5   5 Papule was slightly raised, and was discoloured after the third day.
4	W. B. Male, <i>act.</i> 26. Phthisis. Onset with cough in December 1920. Had a slight hæmoptysis in 1922. Night sweats from December 1923. Had extensive signs in both lungs, was febrile and losing weight. Died a month after the test. Post-mortem examination showed acute miliary tuberculosis. Sputum positive.	6   8   8   6   6   6   6 Papule was slightly raised, and was discoloured on the last day.
29	E. H. Female, <i>act.</i> 17. Phthisis. Onset four years ago. Was sent to a sanatorium and remained well until July 1922. Had signs at the left apex. She became febrile and in November 1922 artificial pneumothorax was started. In September 1924 she had continued pyrexia and extensive disease in both lungs. Sputum positive. Discharged from hospital as a hopeless case.	0   9   9   9   7.5   5.5   5.5 Papule was very slightly raised, and was discoloured after the fifth day.
59	E. B. Male, <i>act.</i> 31. Tuberculous laryngitis diagnosed in August 1924. No pyrexia or loss of weight. Goes about. Swab from larynx showed tubercle bacilli. No signs in lungs, but X-ray showed opacities in both lungs. Two months after the test he had diarrhoea, signs at the right base, pyrexia and progressive loss of weight.	5   8   8 Papule was very slightly raised, and faded completely after the third day.
74	E. T. Male, <i>act.</i> 48. Phthisis. He was first seen in August 1924 with cough, dyspnoea, night sweats and pyrexia. Pleural effusion was found on left side. After aspiration crepitations were detected all over the left lung. Sputum negative. He continued to go downhill, and died with meningeal symptoms towards the end. There was no post-mortem examination.	0   0   3   3   3   3   3 Papule was slightly raised.
85	M. R. Female, <i>act.</i> 17. Acute pneumonic phthisis. Onset in November 1924. Sputum positive. Continued pyrexia and wasting. At first disease was confined to right lung, later it spread to the left.	4.5   4   3 Papule was slightly raised, and faded completely after the third day.

was of type "B," and in one (No. 74) the size of the papule remained the same during the period of observation. The papules were small, only slightly elevated, and showed a tendency to subside or become discoloured towards the latter half of the period of observation. It appears that poor reactivity is associated with extensive or acute infection when the powers of resistance become enfeebled.

It will be interesting in this connection to follow up the course taken by the following four cases (Table VII). Three of these had papules, which, though fairly large, became completely or almost completely discoloured after the second or third day.

TABLE VII.

Case No.	Disease and condition of patient.	Measurements of papule in mm. on successive days (length of one diameter is shown).
102	A. W. Male, <i>et.</i> 49. Phthisis. Tuberculous laryngitis was diagnosed in February 1922. In March 1925 phthisis was diagnosed; he had lost one stone in weight in preceding five weeks. Has a slight evening rise of temperature at times. Physical signs present at both apices. Sputum positive. Later report says weight is rising.	18.5   18.5   17.5   16.5   15.5   13.5 Papule was well raised, but began to fade on the third day and was almost discoloured on the last three days.
103	W. N. Male, <i>et.</i> 29. Phthisis. Five years ago he had pleural effusion on right side. In March 1925 phthisis was diagnosed. There are signs at right apex and base. Weight rising and falling. X-ray shows extensive disease in right lung and discrete mottling in left. Is febrile. Sputum positive. Later report says weight is still rising and falling.	8   7.5   7.5   7.5   7.5   7.5 Papule was slightly raised.
105	F. N. Male, <i>et.</i> 42. Phthisis. Has had cough for two years. Phthisis diagnosed in January 1925. Has signs at right apex, but x-ray shows extensive disease both lungs. Is febrile and is losing weight. Sputum positive. Later reported to be still losing weight.	12   11.5   11   9.5   9.5   9 Papule was moderately raised, but began to fade on the third day and was almost discoloured after that day.
108	E. P. Female, <i>et.</i> 38. Phthisis. Had dry pleurisy in summer of 1922, hæmoptysis in October 1923. Hoarseness for the last six months. In March 1925 phthisis and tuberculous laryngitis were diagnosed. Has signs at both apices. Slight rise of temperature at times. She thinks she has lost weight recently. Sputum positive. Later reported to be progressively losing weight.	13.5   12.5 Papule was moderately raised, but faded completely after the second day.

## BOVINE TUBERCULIN

In the first 21 cases only human tuberculin was used; the remaining 89 cases, which included 36 controls and 53 tuberculous patients, were tested with both human and bovine tuberculins.

Of the 36 control cases, 9, which gave a negative result with human tuberculin, were also negative with the bovine, and 24 showed the type "B" reaction with both tuberculins. In the remaining 3 cases human tuberculin produced the type "B" reaction, while the bovine gave the type "A." Thus the two tuberculins gave identical results in 33 out of 36 cases.

Of the 53 tuberculous patients, 45 gave similar results with the two tuberculins. In 3 cases in which the human gave the type "A" reaction, the bovine produced the type "B." In 4 patients the reverse was the case; and in one instance human tuberculin showed the type "B" reaction, while the bovine gave a negative result. The differences in these 8 cases are suggestive, but a larger series of tests is necessary before any significance can be attached to them.

## SUMMARY

(1) The cutaneous tuberculin test gave a positive reaction in 80·8 per cent. of control cases and in every tuberculous patient tested. The technique of the test has been described.

(2) Two main types of positive reaction can be distinguished :—

(a) The type "A," in which the papule reaches its maximum size on the day of its first appearance. This type of reaction was given by 65·5 per cent. of tuberculous patients, and 94·7 per cent. of these had more or less definite signs of tuberculous toxæmia.

(b) The type "B," in which the papule attains the maximum one or more days after its first appearance. This type was given by the majority of control cases, and 31 per cent. of the tuberculous patients. Of the latter 61·1 per cent. were either cases of chronic arrested disease, or sanatorium patients who had responded well to treatment, had no signs of toxæmia, and were able to work several hours a day.

(3) Tuberculous patients suffering from extensive or acute disease in whom the prognosis is grave gave papules of a nature suggesting poor reactivity. The test is of value in the prognosis of such cases. The features of the reaction in these patients have been described.

(4) In patients in whom the diagnosis of the tuberculous nature of the disease is doubtful the presence of the type "A" reaction is a useful aid in diagnosis, but type "B" does not exclude tuberculous disease.

(5) The results with human and bovine tuberculins were similar in 33 out of 36 control cases, and in 45 out of 53 tuberculous patients.

I wish to express my best thanks to Dr. A. F. Hurst, Dr. G. Marshall, and Dr. A. Neville Cox for the great help they have given me in carrying out these investigations.

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## TWO CASES OF DIVERTICULA FROM THE LOWER END OF THE ŒSOPHAGUS

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital.

THE most common seat for a pouch to develop in connection with the œsophagus is at its extreme upper end, where a weak point is present at its junction with the pharynx. This condition is more correctly described as a pharyngo-œsophageal than as an œsophageal pouch.

The only true œsophageal pouches of clinical importance are those which in very rare cases develop immediately above the diaphragm, generally in front and to the right of the œsophagus. The earliest description of this condition was that of a post-mortem specimen recorded by de Guise in 1833. Eight more post-mortem specimens were described up to 1900. In 1892 Bordoni had diagnosed a diverticulum of the lower end of the œsophagus from a consideration of the symptoms and some not very conclusive tests with a sound, and Minz, Reichmann and Kelling described similar cases.

In 1898 the first case recognised with the x-rays was published by Reitzenstein; since that date only about fourteen additional cases have been described. In the two cases reported by Bensaude and his colleagues (Fig. 1) the condition was also investigated with the œsophagoscope.

The large majority of the cases have been in men. In some no symptoms have resulted. In others the patient has complained of a sense of painful pressure behind the lower end of the sternum during and immediately after meals, dysphagia, regurgitation of food, which had sometimes been eaten long before, and occasionally cough.

The diverticulum is generally lined with pavement epithelium and surrounded by fibrous tissues, no muscle fibres being present.

Two cases of the kind have come under my observation. In neither instance were any symptoms present which could be attributed to the diverticulum. In the first the pouch occurred as an independent condition, as in nearly all the recorded cases. In the second it was associated with achalasia of the cardiac sphincter; similar cases have only been described by Kraus, Vinson and Dessecker.

*Case 1. Epiphrenic Diverticulum of Œsophagus.*—A man of 53 was admitted into New Lodge Clinic for general weakness, accompanied by a feeling of weight in the stomach and flatulence immediately after meals. He had never suffered from dysphagia or vomiting. No evidence of organic disease was found, except that the x-rays revealed the presence of a large diverticulum from the right of the œsophagus immediately above the diaphragm (Fig. 2).

## 362 TWO CASES OF DIVERTICULA FROM THE

*Case 2. Achalasia of the Cardia with a secondary Œsophageal Pouch.*—Pte. C., 33, was taken prisoner on August 27, 1914, in the retreat from Mons after being wounded in the foot. For five days he was almost starved, and after reaching Sinnelager he had bad and insufficient food. Being a Dublin Fusilier, he was sent to Limberg with other Irish prisoners on December 22, 1914, where he refused to join Casement's brigade. He was then treated still more harshly and for five months was not



FIG. 1.

Diverticulum of lower end of œsophagus ( $\times \frac{2}{3}$ .) (Dr. R. Bensaude.)



FIG. 2.

Diverticulum of lower end of œsophagus filled with opaque food, with air above it. ( $\times \frac{1}{2}$ .) (Dr. P. J. Briggs.)

allowed to receive parcels from England. In March, 1915, he began to vomit, and by the end of a month he could keep no solid food down. He rapidly lost weight and strength. At first he kept himself alive with porridge made from oats sent from England, but in the summer he could make no further use of the oats, as no fires were allowed for cooking. On June 1, 1916, he was sent to Switzerland in an extremely emaciated and feeble condition. He was carefully dieted, and gained weight and strength, but was still unable to take any solid food. On June 28 he was given a test-meal; an hour later

almost all the food mixed with much watery mucus was removed. It contained no trace of hydrochloric acid or peptone, but some lactic acid, and as the tube was arrested 41 cm. from the teeth, Dr. Raoux concluded that no food passed beyond the cardia, a view which was confirmed a few days later by an x-ray examination. In September mental symptoms appeared: he had ideas of persecution, and said he wished to commit suicide. He was sent to a mental sanatorium, and on December 21, 1916, he was repatriated. On reaching



FIG. 3.

Diverticulum of lower end of dilated oesophagus: oblique view. ( $\times \frac{2}{3}$ .)  
(Dr. J. R. Caldwell.)

England and being admitted into the Royal Victoria Hospital, Netley, under my care, he stated that the insanity was simulated with the object of getting sent home. He had no pain, but he felt that his food did not get beyond a point beneath the lower end of his sternum. A definite area of cutaneous hyperæsthesia was present on the left side extending for three inches from the middle line from the tip of the ensiform cartilage to an inch above the level of the umbilicus.

An x-ray examination by Capt. J. R. Caldwell, R.A.M.C., showed that he had a greatly dilated oesophagus with a large secondary diverticulum two inches above the cardia (Figs. 3



and 4). He had developed the power of emptying his œsophagus at will, and every morning he washed it out by repeated draughts of half a pint of water; a good deal of the previous day's food mixed with large quantities of mucus and saliva was evacuated. A mercury tube dropped through the cardia without difficulty on January 18, proving the condition to be due to absence of relaxation, or achalasia, and not spasm. The obstruction rapidly diminished as a result of the daily passage of the mercury

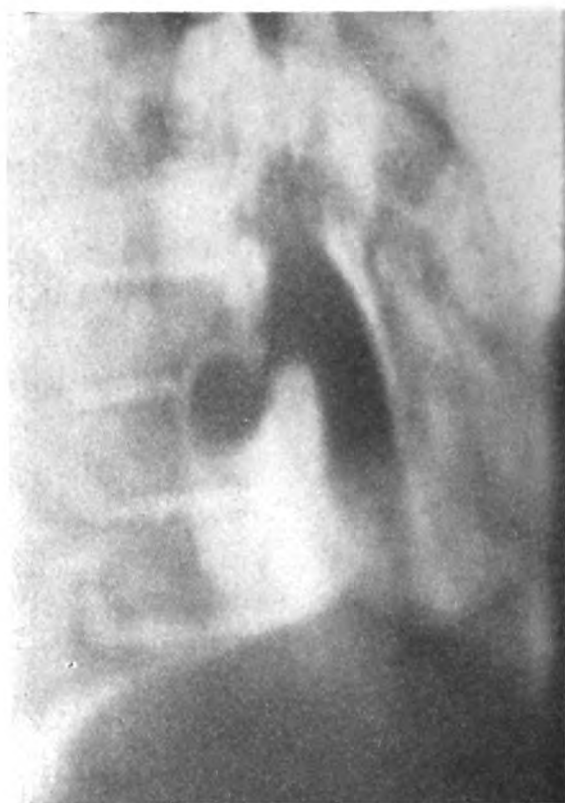


FIG. 4.

Same case as fig. 3, after vomiting; the diverticulum is now more clearly seen. ( $\times \frac{2}{3}$ .) (Dr. J. R. Caldwell.)

tube by the patient himself. When I last heard of him, in 1920, he could eat solid food without difficulty and only passed his mercury tube about once a week.

The following weights show the result of treatment :—

	Stones.	Pounds.
On mobilisation : August, 1914 . . . . .	9	8
On reaching Switzerland, June, 1916 . . . . .	7	4
On reaching England, December 29, 1916 . . . . .	8	3
On first passage of tube, January 17, 1917 . . . . .	8	7
On January 24 . . . . .	8	11
On January 28 . . . . .	9	1
On February 11 . . . . .	9	7
On March 10, 1917 . . . . .	9	12

*Other Recorded Cases*

Of the four cases in the literature, in which a dilated œsophagus secondary to achalasia of the cardia was complicated

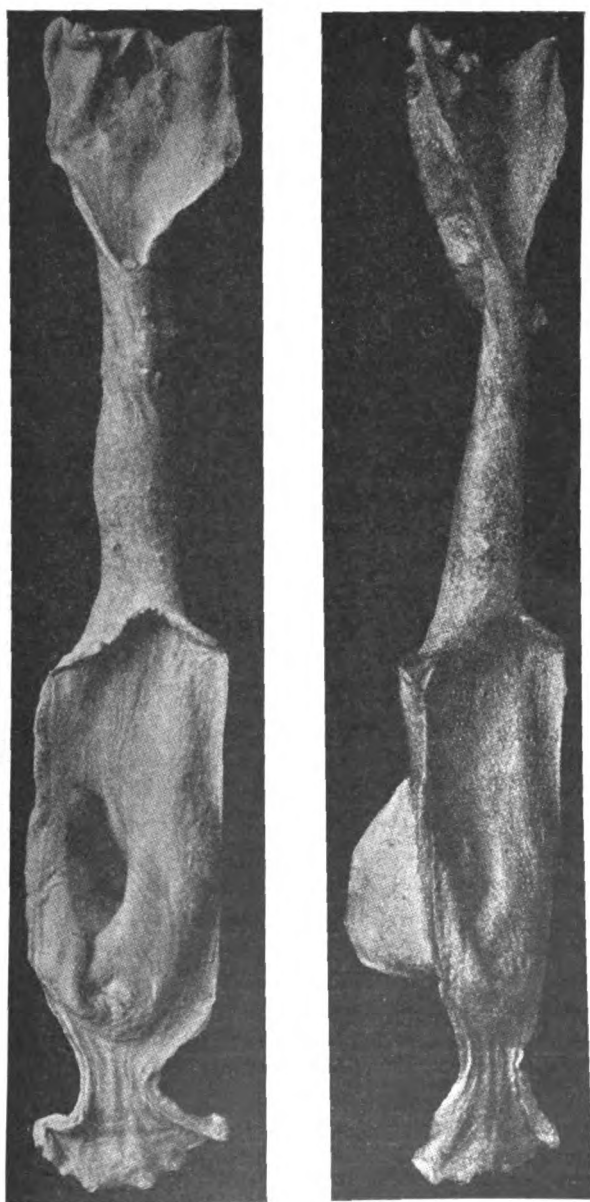


FIG. 5.

Diverticulum of lower end of dilated and hypertrophied œsophagus. (Kraus.)

by the presence of a diverticulum, one was only found post-mortem; the other three were diagnosed with the x-rays.

Kraus in 1902 described a specimen in which a diverticulum had formed from the left side of a dilated œsophagus just above the diaphragm; no organic obstruction of the cardia was present (Fig. 5). The mouth of the diverticulum formed a gap of 6 cm. long and 3.5 cm. wide in the greatly hypertrophied muscular coat of the œsophagus. The wall of the diverticulum itself consisted of mucous membrane surrounded by fibrous tissue with no muscle fibres.

In 1923 Vinson reported two cases from the Mayo Clinic, in which the x-rays showed the presence of a pouch arising from the left side of the middle of the œsophagus, which was dilated as a result of achalasia of the cardiac sphincter. In neither case had the pouch given rise to symptoms.

In 1924 Dessecker described a case in which a dilated œsophagus was associated with a diverticulum on the right side just above the diaphragm. The patient complained of attacks of pain after food very high in the epigastrium.

In these cases the pouch probably developed as a result of the increased pressure in the lower end of the œsophagus caused by violent peristalsis attempting to overcome the obstruction produced by achalasia of the cardia<sup>7</sup> in the early stages before much dilatation had occurred. There was presumably a congenitally weak point in the wall of the œsophagus where the pouch developed. In some of the cases the weakness may have been secondary to old mediastinal disease, such as an inflamed or tuberculous mediastinal gland, which had become adherent to the œsophagus and led to a minute traction diverticulum; in its turn the stasis of food in the dilated œsophagus caused by the achalasia would lead to great enlargement of the original traction diverticulum. In such cases the achalasia itself may have been a reflex result of irritation caused by the adhesion to the lower end of the œsophagus.

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## ANTERIOR PHARYNGO-ŒSOPHAGEAL POUCH AS A CAUSE OF DYSPHAGIA

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital, and  
P. J. BRIGGS, M.A., Radiologist to New Lodge Clinic.

ALL cases of dysphagia can be roughly grouped into three classes: in the first the difficulty is experienced in passing from the pharynx into the œsophagus; in the second the difficulty occurs somewhere in the course of the œsophagus, most frequently about the level of the bifurcation of the trachea; in the third the difficulty is in the passage from the œsophagus into the stomach. In the first class the common causes are malignant disease and paralysis, generally organic, but occasionally hysterical, and, much less frequently, a pharyngeal pouch; in the second class malignant disease is nearly always present; in the third class the cause is either achalasia of the cardiac sphincter or cancer involving the lower end of the œsophagus or upper end of the stomach.

In the course of years I have seen about half a dozen cases of the first class, in which no cause could be discovered for the difficulty experienced in the initiation of the act of deglutition, when the bolus of food is transferred by a voluntary act from the back of the tongue, through the pharynx, into the upper segment of the œsophagus. No abnormality was found with the x-rays or the œsophagoscope; no sign of organic nervous disease was discovered, and in spite of a suspicion that an early growth or a very early stage of bulbar paralysis of some kind might have been missed, the dysphagia, which was always paroxysmal in nature and often accompanied by distressing attacks of choking, was finally regarded as hysterical. I find that other physicians and laryngologists have come across similar cases.

The history of the following case is typical of the group to which I have just referred. I have recorded it in detail, because I think that the cause of the dysphagia which was finally discovered could be so easily overlooked, that it may well be the cause also of some at any rate of the other cases with a similar history in which no other explanation is found.

### *Dysphagia due to Anterior Pharyngo-Œsophageal Pouch*

An unmarried lady, aged 57, with no worries and no previous illnesses, interesting herself in literature and parish work, was

admitted to New Lodge Clinic on April 21, 1925. In September 1923 she had for the first time experienced an aching sensation behind the upper end of the sternum. The next month, whilst at lunch, she suddenly felt difficulty in swallowing some water. She managed to get it down, but ever since then the same thing has been liable to occur two or three times a week, though occasionally two or three weeks pass without an attack. Sometimes the food appears simply to stick for a moment and then pass on; at other times it regurgitates into her mouth, often with the addition of a little stringy mucus. Liquids are as



FIG. 1.

Anterior pharyngo-oesophageal pouch seen from the front. The shadow at the top is the jaw, slightly tilted to one side. The median shadow is the pouch filled with opaque food, a thin stream of which can be seen passing in a curved direction down the extreme outer sides of the commencement of the œsophagus. (Full size.) (Dr. P. J. Briggs.)

likely to give rise to trouble as solids, and soft food more than hard. When the food has either passed on or been regurgitated, she can finish a meal without difficulty. The obstruction occurs much more commonly at breakfast than at other meals; the aching behind the upper end of the sternum, which often precedes the actual obstruction, may persist all day, but the difficulty in swallowing is rarely repeated.

Since the onset of the dysphagia she has lost about 10 lbs. in weight, though her appetite remains fairly good.

The patient had been examined by a laryngologist and on

more than one occasion by a radiologist, but no cause of the dysphagia was discovered.

From a consideration of her symptoms it seemed clear that the difficulty in swallowing was due to some impediment at the junction between the pharynx and œsophagus. Ordinary physical examination failed to reveal any abnormality. The patient's nervous system seemed to be absolutely normal, and there was nothing pointing to peripheral neuritis, myasthenia gravis, bulbar paralysis or a primary myopathy. If a growth



FIG. 2.

Anterior pharyngo-œsophageal pouch seen from the side. ( $\times \frac{3}{4}$ .)  
(Dr. P. J. Briggs.)

were present it would have been visible with the laryngoscope and easily palpable after such a long period, but nothing abnormal could be felt or seen.

When the act of swallowing was watched with the x-rays, the first mouthful of the opaque meal was seen to be arrested in a small pouch in the middle line immediately in front of the entrance of the œsophagus and behind the larynx (Figs. 1, 2 and 3), although at the time there was no dysphagia. The pouch extended backwards on filling, apparently because the larynx prevented it from pushing forwards; the centre of the œsophageal mouth was thus obstructed, and the rest of the opaque meal was seen to pass down as a thin stream on each side of

the pouch, the two streams meeting below and then passing down the œsophagus in the normal way.

After the pouch had been discovered with the x-rays, Mr. Gill-Carey passed a short œsophagoscope under a local anæsthetic after the injection of morphine and scopolamine. It entered the œsophagus without difficulty, and no abnormality was at first seen. The tube was then slowly withdrawn, and a transverse slit was discovered immediately behind the



FIG. 3.

Anterior pharyngo-œsophageal pouch seen obliquely, so that one of the thin lateral streams of opaque food appears to be in front of the pouch. ( $\times \frac{2}{3}$ .) (Dr. P. J. Briggs.)

arytenoid cartilage and in front of the mouth of the œsophagus. A fine bougie was passed into this, the slit representing the opening into the pouch, which appeared to be about three-quarters of an inch in depth.

The patient accurately localised the seat of obstruction, and when dysphagia occurred she had pain in this situation, but the more common sense of discomfort which she has experienced was for some obscure reason felt behind the upper part of the sternum.

As no serious symptoms had resulted from the dysphagia,

no operation was advised. The nature of the condition was explained to the patient. She was advised to try to cough up anything which stuck in her throat at once, and to drink some water five minutes after the completion of each meal in order to wash out any food residue. She was also advised to avoid swallowing any pips or skins of fruit or anything else which had not been chewed to a fluid consistence in order to prevent fragments from lodging in the pouch and leading to its inflammation.

A study of Fig. 4, taken from Cunningham's *Anatomy*, affords an anatomical explanation for the occurrence of an anterior pharyngo-œsophageal pouch. It probably forms where the two longitudinal bands, which pass from the front of the œsophagus to end in a tendinous prolongation which is attached to the back of the cricoid cartilage, emerge above the uppermost circular fibres of the œsophagus and are slightly separated from each other so that a weak point is left. Through this a pouch of mucous membrane may be forced.

An anterior pouch formed in this way cannot press forwards when filled owing to the resistance offered by the larynx; it therefore expands backwards and obstructs the entrance into the œsophagus. Normally food can be seen with the x-rays to be shot downwards in the middle line from the back of the tongue into the cervical portion of the œsophagus. In our patient the first mouthful was arrested by entering the pouch. The rest of the food was then seen to pass in two streams down the extreme lateral walls of the pharyngo-œsophageal junction, the passage down the centre being obstructed by the filled pouch. When all the food had been swallowed the pouch was slowly emptied, presumably as a result of external muscular pressure, as it is very improbable that it has any muscular fibres of its own.

Simple filling of the pouch does not cause dysphagia, as the patient did not experience any difficulty in swallowing or feel any local discomfort on the three occasions on which she was examined with the x-rays. Presumably her paroxysmal attacks are due to occasional over-distension of the pouch, which produces complete obstruction, so that any additional food is regurgitated. Relief occurs when the contents of the distended diverticulum are ejected.

It is easy to understand why this condition has escaped observation in the past. In the routine x-ray examination of the œsophagus the first stage of deglutition is very rarely watched, the radiographer concentrating his attention on the thoracic œsophagus; when there is a history of dysphagia



affecting the upper part of the œsophagus, the cervical œsophagus is watched and the well-recognised lateral pouches are looked for, but the actual process of propelling the bolus through the pharynx is rarely observed.

Our patient had been previously examined by one of the most experienced and careful radiographers in the country without anything abnormal being noted, and it was only when special attention was paid to the pharyngo-œsophageal junction that the pouch was discovered. It was also quite conceivable

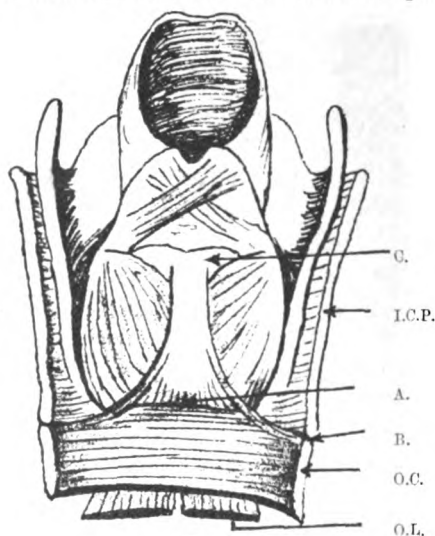


FIG. 4.

Dissection of pharyngo-œsophageal junction from behind with mucous membrane removed (from Cunningham's *Anatomy*). O.C., circular fibres of œsophagus; O.L., longitudinal bands from œsophagus united by fibrous elongation to cricoid, C.; I.C.P., inferior constrictor of pharynx. A is the weak point through which the anterior pouch forms. B is the gap between the inferior constrictor of the pharynx and the circular fibres of the œsophagus; the more common lateral pouches generally form in the mid-line behind, where there is also a gap in the longitudinal fibres, and then pass to one side.

that the pouch does not always get filled during a meal. In our case, however, it was filled on every occasion that the examination was made although no dysphagia was present at the time.

It is equally easy to miss the pouch with the œsophagoscope, as it passes without obstruction into the upper end of the œsophagus. Only when it is withdrawn so that the posterior wall of the glottis and the mouth of the œsophagus come under observation can a transverse slit be seen. This looked like a simple fold of mucous membrane, and it was only found to be the opening of the pouch when the end of a curved bougie was passed three-quarters of an inch into it before being arrested by meeting its lower extremity.

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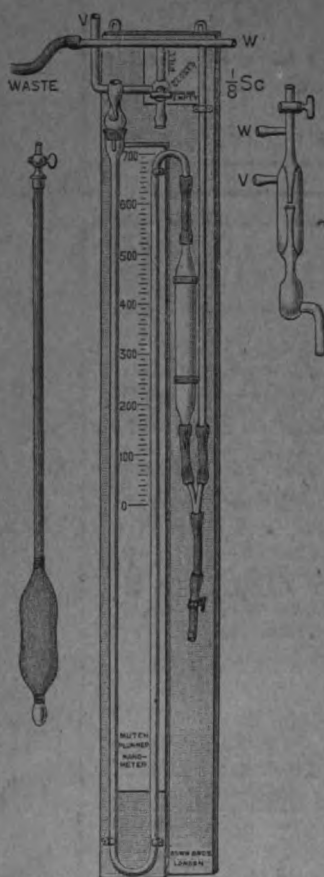
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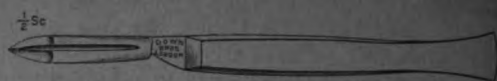
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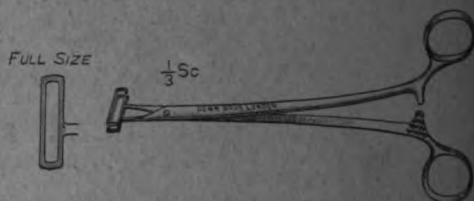


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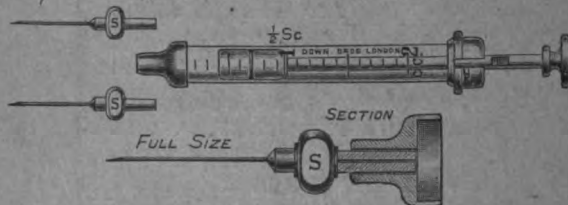
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## ALEXANDER JOHN GASPARD MARCET

PHYSICIAN TO GUY'S HOSPITAL, 1804-1819

By SIR ARCHIBALD GARROD, K.C.M.G., M.D., F.R.S., Regius Professor  
of Medicine in the University of Oxford; Consulting  
Physician to St. Bartholomew's Hospital.

THE tenets of the French Revolution met with ready acceptance in the neighbouring Republic of Geneva. Throughout the eighteenth century that little state had been the seat of recurrent political struggles, such as are inevitable in a community in which all power is in the hands of the members of a limited number of families, whereas the far more numerous descendants of more recent settlers are excluded from the franchise. In those struggles now one side and now the other had gained the advantage; but in the end the oligarchy had retained its power, by outside aid from France or from some Swiss Cantons.

The course of events in Geneva conformed to the French pattern. A popular uprising, followed by overthrow of the oligarchy; the setting up of a Constituent Assembly, which framed a wholly democratic constitution; oaths of fraternity and trees of liberty.

Thereafter, in 1794, a further uprising, fomented by the French Representative; mob rule; wholesale arrests, by the self-styled "Montagnards," of members of the patrician families, and a Revolutionary tribunal. The short-lived "reign of terror" which ensued was far less bloodstained than its prototype in Paris; and although some of the prisoners were condemned and shot, they were, for the most part, either released, fined or banished.

Amongst them were two young men, friends from childhood and members of good families, Alexander John Gaspard Marcet and Gaspard Charles de la Rive, who during the months of their imprisonment determined that, if released, they would make their way to Edinburgh and would there study medicine.

Marcet, who was born in 1770, was the son of a Genevan merchant, of Huguenot descent, a member of an old family, to which the Emperor Charles V had granted armorial bearings and the right to quarter the arms of St. André. His father, on his death-bed, had urged him to follow in his own footsteps, and to



take up a mercantile career. This injunction the son followed loyally, but after two years, which served only to confirm his dislike of commerce, he abandoned it in 1790, in favour of the study of law.

In 1793, during the earlier stage of the Genevan revolution, he had travelled to England, in company with Nicholas Theodore de Saussure, the elder son of the celebrated Horace Benedick de Saussure, who himself attained to considerable scientific eminence. It was shortly after their return, in the following year, that Marcet was arrested, on the pretext that he had been an officer in the National Militia. Eventually, both Marcet and his friend de la Rive were released from prison, but banished for five years; and in accordance with their plan they came to Edinburgh in October 1794, and matriculated at the University as students in the Faculty of Medicine.

Undoubtedly Marcet found his true vocation, at last, when he entered upon the study of science and medicine; and the University of his choice was celebrated throughout Europe as one of the great centres of such studies. At that time the illustrious Joseph Black still occupied the Chair of Medicine and Chemistry, and the chemical aspects of medicine were attracting much attention in the University. Although Bransby Cooper mentions Black amongst Marcet's teachers, their association can hardly have been close, for when, in the year of his graduation, Marcet was asked to obtain some information from Black, he approached him through another professor. It was to Daniel Rutherford, Professor of Botany and Clinical Medicine, and Cullen's successor as Physician to the Royal Infirmary, that Marcet dedicated his Thesis for the doctorate, as well as to his own brother-in-law, Professor Prevost, Rector of the University of Geneva. Rutherford was a pupil of Cullen and Black and was deeply interested in chemistry, and indeed it was he who first distinguished clearly between nitrogen and carbonic acid gas. Also among Marcet's teachers was James Gregory the younger, a member of a family which had produced more professors than any other; the successor of Cullen in the Chair of the Practice of Physic. Of him Christison wrote that he was the most captivating lecturer whom he had ever heard, and his name is immortalised in connexion with rhubarb and magnesia.

In due course, in 1797, the two friends took their degrees, as Doctors of Medicine, on the same day. Marcet took "Diabetes" as the subject of his thesis, and de la Rive "Animal Heat."

The opening sentences of Marcet's thesis sound the key-note of his life's work: "*Chemia nuperrime tantum in physiologicam*

investigationem, felici ullo successu, introduci potuit. Utilissima hæc scientia empirico tantum, et quasi cæco auxilio medicinam olim adjuvabat, et theoria ad nova experimenta suscitanda nihil fere conferebat. Quantum vero, his diebus, lucis et utilitatis medicina a chemicis indagationibus accipere possit, morbus de quo in hoc testamine agitur, insigne exemplum præbere mihi videtur."

Their term of banishment had still two years to run, and both the young men came to London, where, in 1799, de la Rive became an Extra Licentiate, and Marcet a Licentiate of the Royal College of Physicians. When free to do so, de la Rive determined to return to Geneva, which had, in the meantime, been annexed to France as chief town of the Department of Leman. Marcet, on the other hand, resolved to remain in England, and to practise as a physician in London; but it is clear that before doing so he paid a visit to his native place, for in a paper read before the Medical Society in 1801, he spoke of having been in Geneva about twelve months previously. In 1800 he was naturalised as a British subject by Act of Parliament.

His first London residence was in the neighbourhood of St. Mary Axe, where lived friends who had mercantile connexions with Geneva, and among them a wealthy merchant, of Swiss descent, Mr. Francis Haldimand, to whose daughter, Jane, afterwards to become a celebrated authoress, Marcet was married in 1799.

In addition to Mrs. Marcet, two other members of her family find places in the *Dictionary of National Biography*, namely, her great-uncle, Sir Frederick Haldimand, who became Governor of Canada, and her brother, who was a Governor of the Bank of England and Member of Parliament for Ipswich.

In London, as in Edinburgh, Marcet was among men of like tastes and interests, the senior members of a line of chemist-physicians who played a prominent part in the advance of British medicine during a large portion of the nineteenth century. With two of them, both men of brilliant ability, Marcet was associated in many investigations, namely William Hyde Wollaston and William Prout. Wollaston, although he was only four years older than Marcet, took his M.D. degree at Cambridge before the latter matriculated at Edinburgh; but Prout was a younger man, born in 1785. Marcet had the highest respect for Wollaston's work and opinion; consulted him frequently when confronted with a difficulty; and dedicated to him his book on Calculous Disorders.

The Haldimands were friends and near neighbours of Astley Cooper, who became one of Marcet's closest friends. Others

such were John Yelloly, Physician to the London Hospital, and William Babington, Physician to Guy's.

At that time the Fellowship of the Royal College of Physicians was closed to all, with few exceptions, who were not Doctors of Medicine of Oxford or Cambridge, and Marcet had to be content to remain a licentiate throughout his life. His first appointment was that of Assistant Physician to the Carey Street Dispensary, which was soon followed by that of Physician to the City Dispensary. He attached himself to Guy's Hospital as a pupil, and there, as Nisbet says, "he gained the particular partiality and friendship of Dr. Saunders, an able judge of rising merit and abilities." When, in 1804, a vacancy occurred upon the staff of the hospital, he was advised by Dr. Saunders to stand, and thanks, largely, to the interest and exertions of his teacher, he was elected to the vacant post of Physician. At Guy's, too, he was associated with the Quaker chemist and philanthropist William Allen in the Lectureship of Chemistry, and he continued to lecture on that subject during the whole of his tenure of the physiciancy, and for a year after his resignation from the staff.

It would not appear that private medical practice had any strong attraction for Marcet. Nisbet wrote of him that "his mind is above the bustle and cares of mere professional life, and however zealous in practice, he had no ambition to court popularity." Nevertheless, his clinical work was carried out with his usual thoroughness. The author of the unsigned obituary notice in the *Medical and Physical Journal*, who obviously writes from intimate personal knowledge of Marcet, tells us that "he was in the constant habit of noting down, with great minuteness, the history and daily variations in the symptoms of every case that fell under his observation, and that presented any point of interest, both in his hospital and private practice."

The same writer speaks of "the services which he rendered to the Medical School at Guy's Hospital, by the removal of several obstacles which formerly stood in the way of a principal source of medical knowledge," and of the reforms which he brought about, in the face of much opposition, in the dietary of the patients in the wards. "The success of this measure was highly gratifying to him, and he always regarded it as one of the most useful things that he had ever done."

Chemistry was always Marcet's favourite study. He had fitted up a private laboratory which was, we are told, a model of neatness and order. When the great Swedish chemist Berzelius paid a visit, of some duration, to England in 1812, he and Marcet

worked together on the composition of carbon disulphide, and their results are embodied in a joint paper in the *Philosophical Transactions*. Presumably it was during that visit that they became friends, for it is not recorded that Marcet ever visited Stockholm. He certainly was made a member of the Royal College of Medicine in Stockholm; and it is on record that it was in consequence of Marcet's advice that Berzelius adopted the plan of illustrating his lectures by experiments, with the result that "when he added a series of experiments, easily understood, to his own eloquent words, his course became an object of admiration as well as a model for the other schools of Europe." This being so, we are not surprised to learn that it was also Marcet who "introduced the plan of clinical lectures at Guy's Hospital."

Marcet was elected a Fellow of the Royal Society in 1808, and was also a Fellow of the Geological Society and an active Member of the Royal Institution. In addition to the Swedish College already mentioned, he was a member of the Society of Medicine of Paris, and of the Society of Natural Philosophy and Natural History of Geneva.

With his friend Dr. Yelloly he brought about the foundation, in 1805, of the Medical and Chirurgical Society, the nucleus of the present Royal Society of Medicine, of which he was, from the first, Foreign Secretary, Councillor and Trustee. There was, at that time, widespread dissatisfaction with the management of the older Medical Society of London, and especially with the tendency to indefinite tenure of offices therein.

In the year 1808 Marcet moved to 23 Russell Square, where he had as a near neighbour Sir Samuel Romilly, a distinguished lawyer, of Huguenot descent, who had ties with Geneva, and is entitled to remembrance as the reformer of the Draconic criminal code then in existence. His son, Edward Romilly, afterwards married one of Marcet's daughters.

In the following year, after the ill-starred Walcheren expedition, special measures were needed to cope with the enormous amount of sickness amongst the returning troops. A temporary military hospital was established at Portsmouth, and Marcet, who when additional medical personnel was called for had offered to help, was appointed its Superintendent. The Walcheren fever was the endemic remittent or intermittent fever of marshy countries, followed a seasonal law and was not directly contagious. Borland and Lemprière state that the crews of vessels lying only a few yards from shore, in the narrow channel between the islands of Beveland and Walcheren, remained perfectly healthy throughout the campaign. Yet, after a few months,

Marcet acquired a "similar disease," from which he recovered with difficulty.

Meanwhile Mrs. Marcet was beginning to achieve distinction as a writer of popular books on scientific subjects. In those days such books were rare, and hers were of exceptional merit. Indeed a reader of to-day can easily understand the great success which they obtained, and cannot fail to admire the lucidity with which such subjects as chemistry, physics and political economy are made as clear to young minds as the knowledge of that day allowed.

The *Conversations on Chemistry*, published in 1806, passed through eighteen editions, and no less than 160,000 copies are said to have been sold in the United States alone. *Conversations on Natural Philosophy* reached a fourteenth edition. *Conversations on Political Economy*, her masterpiece, met with cordial commendation from such masters of its subject as Jean Baptiste Say and J. R. McCulloch; and Macaulay wrote of it that "every girl who has read Mrs. Marcet's little dialogues on Political Economy could teach Montagu or Walpole many lessons in finance."

It is not easy, in the absence of contemporary memoirs and letters, to gain a clear picture of the personality of one who lived a hundred years ago, but it would seem evident that Marcet was highly esteemed by his fellows. The following words from an obituary notice ring true: "It is the lot of few to be so much beloved as Dr. Marcet universally was by the profession of which he was one of the most distinguished ornaments," and a more personal note is struck in the following quotation from the biography of Sir Astley Cooper: "His death caused a vacancy in the list of Mr. Cooper's intimate friends which was never afterwards filled up." In the same biography we obtain some interesting glimpses of Marcet, chiefly on his visits to Gadsbridge, the country house near Hemel Hempstead which Cooper had bought. On one occasion, on arriving at Gadsbridge, "Dr. Marcet made his appearance as punctually as at a London party, having given himself just time sufficient to permit him to arrange himself with his accustomed elegant neatness for the dinner-table." This recalls the description of his laboratory as "a model of neatness and order."

On a certain Sunday, also at Gadsbridge, "Sir Astley took Dr. Marcet and Dr. Babington into his dissecting-room, having previously prepared several objects for chemical analysis, and there they remained while the rest of us were at church. Dr. Marcet had brought down a small portable laboratory with him, his constant companion when he visited my uncle at Gadsbridge."

Another story relates to a shooting party at which a fine was to be paid to the keeper by anyone who shot a hen pheasant, and after which Marcet found that a fellow guest had slipped a brace of hens into his bag. "Upon this discovery, Dr. Marcet, a man of high feeling, and one who was most punctilious in the observance of all matters of decorum, became very angry, and did not fail to express, as strongly as he could in his broken English, his indignation at the liberty which had been taken with him"; a story which says more for his sense of propriety than of humour. His published writings show no lack of command of the English tongue.

The author of the biography, Bransby Cooper, also mentions Marcet's "remarkable facility of applying his knowledge to the daily concerns of life" and his delight "in suggesting improvements in matters which might appear almost too trifling to attract his notice," such as a way to de-lead a gun by running mercury up and down the barrel.

We read, elsewhere, of the persuasive suavity of his manners, of his zeal in promoting every object of public utility, and of "the generous ardour of his disposition to excite the zeal of others, and to secure their co-operation in every laudable undertaking."

Despite his acquired British nationality, and his long residence in England, the ties with Geneva were not broken; and in 1815 Marcet revisited his birthplace with his family. Geneva had, by that time, regained its independence, and had entered the Swiss Confederation as a constituent Canton.

Not long after that the death of Mr. Haldimand placed Mrs. Marcet "in possession of an ample fortune," and Marcet resolved to abandon the practice of medicine and to devote himself to scientific research.

Accordingly, in 1819, he resigned the office of Physician to Guy's, but continued to lecture on chemistry there until the following year, when he returned to Geneva for a much longer stay. His native city received him well; he was appointed Honorary Professor of Chemistry in the Academy (University), and was elected a member of the House of Representatives. In his lectures at the University he was once more associated with the friend of his youth and companion of his Edinburgh days, Gaspard de la Rive. In a letter written by his Quaker friend, William Allen, when on a visit to Geneva, we have a glimpse of Marcet at that time (February 1820). Allen wrote: "He lives in the same house, a fine, large building formerly occupied by Theodore de Saussure. Marcet took me to hear a lecture of de la Rive's upon light and electricity—it was a very excellent one.

. . . After the lecture I went to Marcet's to dinner, met Pictet and de la Rive, and we had a very comfortable little party. De la Rive and Marcet had to attend at the House of Representatives, and I walked back to Secheron."

So the old ties were reknit, and we are not surprised that Marcet determined to take up his abode in the land of his birth. With this intention he bought the property of Malagny, in the neighbourhood of Geneva, which had long been in the possession of the Saladin family.

In the autumn of 1821 he returned to England to make arrangements which such a migration entailed. In March 1822 we find him reading his last papers before the Medical and Chirurgical Society, based upon observations made years previously, and in the late summer he carried out a long-cherished plan "of making the tour of Scotland." He returned to London in good health, but shortly afterwards "he was seized, while in the neighbourhood of London, with a sudden attack of gout in the stomach, from the effects of which he had scarcely recovered when a return of the disorder took place and was immediately fatal." His death occurred on October 19th, 1822, in the fifty-third year of his age. Bransby Cooper tells us that he was attended by Astley Cooper and Babington, but gives no name to his illness. The materials do not suffice for a revised diagnosis. The Medical and Chirurgical Society paid founder's honours to his memory. At the next meeting after his death, a committee was appointed to convey to Mrs. Marcet the sympathy of the Society, and the ordinary business of the meeting was postponed to the following week. Not long afterwards his widow presented to the Society the beautiful portrait of her husband by Raeburn, which now adorns the Council Room of the Royal Society of Medicine. The photograph of that portrait here reproduced, by kind permission of the President, is not able to show the most striking feature, the deep blue colour of the eyes.

Mrs. Marcet outlived her husband many years, and died in 1858, at the age of eighty-nine. Their son, Francis Marcet, although British by birth, was Swiss by adoption. He lived chiefly at Malagny, was a Member of the Legislature of the Canton of Geneva, and an Honorary Professor in its University. He was a distinguished physicist and vegetable physiologist, and, like his father, was a Fellow of the Royal Society. The son of Francis, William Marcet, was a Doctor of Medicine of Edinburgh, a Fellow of the Royal Society and of the Royal College of Physicians, and was, for a time, Assistant Physician to the Westminster Hospital. His researches on respiration, which

are embodied in his Croonian lectures, have secured for him a place of honour in the records of British medicine. His only son, Alexander Marcet, the last of the name, shared to the full the scientific interests of his forebears. He became a civil engineer, but died at the early age of forty-three. Truly an interesting example of inherited scientific ability, in the descendants of a highly gifted couple.

Alexander Marcet's published writings constitute his monument, and these fall into several groups. Some of his papers treat of purely chemical or physico-chemical subjects; others are records of clinical cases; others, again, deal with bio-chemical subjects, some physiological and some pathological. Lastly, some articles deal with matters of more general interest, such as the secondary schools at Geneva, and the Hospice de la Maternité in Paris.

There can be no doubt that Marcet had a very thorough chemical training, and as a chemist he was certainly no amateur. He is said to have been "particularly distinguished by his skill in analytical researches and his extreme precision in the mode of conducting them."

Some of his minor contributions bear witness to the ingenuity of which Bransby Cooper speaks. Thus in a letter to Edward Jenner (*London Medical and Physical Journal*, vol. ix. p. 462, 1803), he suggests a device for the preservation of vaccine lymph, in a chamber formed by polishing a central area in two small plates of ground glass. When the ground surfaces are in contact the cavity is air-tight. Again (in Nicholson's *Journal of Natural Philosophy*, etc., vol. xxxiv. p. 119, 1813) he describes the freezing of mercury by evaporation of ether under the air-pump, and later, the still greater efficiency of carbon disulphide for this purpose. Elsewhere we find a method for the production of "intense heat" by "urging the flame of a spirit lamp by a current of oxygen gas" (Thomson's *Annals of Philosophy*, vol. ii., p. 99, 1813), by which means he was able to burn a diamond. The oxyhydrogen blowpipe had been invented some ten years earlier and the credit thereof is shared between Thomas Thomson and Hare.

Marcet evidently took much interest in the analysis of waters and published analyses of the Brighton chalybeate spring and of one near Niton, Isle of Wight. Other papers deal with the waters of the Dead Sea and River Jordan. To the *Philosophical Transactions*, Pt. 2, p. 161, 1819, he contributed a paper of no less than forty pages, on the temperature, specific gravity and saline content of the water of particular seas and lakes. This



work, carried out upon samples brought from all parts of the world, must have entailed much labour, and led to some interesting conclusions.

In their conjoint paper Berzelius and Marcet (*Phil. Trans.*, p. 171, 1813) described their investigation of the substance then called alcohol of sulphur, but now carbon disulphide. The views then held as to its nature, and even its constituents, differed widely. The authors showed that it contained only carbon and sulphur in the proportion of one part to two. After their work was done, but before it was published, Berthollet, Thenard and Vauquelin published results which were practically identical. Marcet was also responsible for the articles "Potassium" and "Platina" in Rees' *Encyclopædia*.

Marcet's clinical papers deal, for the most part, with individual cases which illustrate special points or show exceptional features; but it is difficult to project one's mind backwards to the state of clinical knowledge a hundred years ago, and to attempt to estimate the novelty of facts and views brought forward in writings of that date.

In the introduction to a paper in which he gave a detailed account of a case of hydrophobia, and of the autopsy (*Med. Chir. Trans.*, vol. i. p. 133, 1811) Marcet points out that only by comparing cases of a malady can its diagnostic features be recognised; and adds that "it is from accuracy of detail that an insulated case derives its principal interest, and that circumstantial accounts afford the only reasonable ground for future generalizations." He calls attention to the pain felt in the region of the bite, which has been noted in cases of hydrophobia, and which follows the course of nerves and not of lymphatics; and suggests that when excision or cautery has been omitted at the onset, it may still be useful at any later time before the disease develops.

Marcet's graduation thesis on diabetes has already been mentioned, and his first clinical paper dealt with a case of that disease (*London Med. and Phys. Journ.*, vol. ii. p. 209, 1799) in which advanced pulmonary tuberculosis, which had not been diagnosed during life, was found *post mortem*. It is noted that the pancreas was paler and firmer than usual.

In 1805 he recorded the case of a girl aged twenty-one admitted to Guy's Hospital with extreme cyanosis and œdema of the limbs, which led those who saw her to suspect a grave cardiac lesion (*Edinburgh Med. and Surg. Journ.*, vol. i. p. 412, 1805). The cyanosis was of seven weeks' standing. The patient died soon after her admission, and at the autopsy no cardiac lesion nor defect was found; the heart was somewhat

enlarged, but there were no pericardial adhesions. The lungs were everywhere adherent, to parietes, diaphragm and pericardium. Some of the adhesions were of long standing, but there were "large quantities of coagulable lymph"; the lungs were engorged but floated on water.

It was noted, as unusual, that the blue colour faded soon after death, and by the next day had almost disappeared. Presumably the case would now be described as one of mediastinitis. In the discussion no stress is laid upon the œdema; it was pointed out that patency of the foramen ovale does not necessarily give rise to cyanosis, and conversely, that extreme cyanosis may be due to other causes than septum defects.

Another paper describes the recovery of a youth who had swallowed no less than six ounces of laudanum (*Med. Chir. Trans.*, vol. i. p. 77, 1809). Despite the fact that treatment was only begun five hours later, his life was saved by emesis and constant movements.

In 1810 Marcet gave an account of a severe case of erythema, not due to mercury (*Med. Chir. Trans.*, vol. ii. p. 73, 1810). Such exfoliative dermatitis, as we should call it, had been described by Spens and Alley as a result of mercurial treatment, and named "hydrargyria," but Marcet concludes that such eruptions may result from various causes, and that Alley's name was not more appropriate than would be that of "plumbalgia" for intestinal colic.

A paper on a case of calculous nephritis (*Med. Chir. Trans.*, vol. x. p. 147, 1819) gives a vivid picture, in the patient's own words, of the pains and discomforts which he endured, and his experience of lithotomy without an anæsthetic.

The last of all the papers (*Med. Chir. Trans.*, vol. xii. p. 52, 1822) records, as a medical curiosity, the case of a sailor who, in drunken bravado, swallowed several clasp-knives, and who repeated the feat at intervals during the following ten years. What happened to the knives, thirty-five in all, and how they brought about his death, is told in detail. The specimens are to be seen in the Museum of Guy's Hospital.

Two earlier contributions treat of the use of bismuth subnitrate (magistery of bismuth) in the treatment of dyspepsia, and of extract of stramonium in sciatica and other painful affections.

More important than these purely clinical writings are those which may be classed as bio-chemical. A group of papers dealing with the chemical composition of the body fluids, taken in conjunction with those of John Bostock and others upon the same subject, laid the foundations of our present knowledge.

They include the earliest analyses of cerebro-spinal fluid, as obtained from cases of spina bifida and chronic hydrocephalus, and independent analyses of various dropsical fluids (*Med. Chir. Trans.*, vol. ii. p. 342, 1811).

At that time the alkalinity of the blood and body fluids was thought to be due to caustic alkali, and not to bicarbonate. George Pearson maintained, in opposition to other investigators, that the free alkali of the blood was potash, and the publication of Marcet's results, which disposed of that view, started a controversy between the two which on Marcet's side was conducted with polite dignity, but on Pearson's was not. To the same group belongs a paper entitled: "Some Experiments on the Chemical Nature of Chyle, with a few Observations on Chyme" (*Med. Chir. Trans.*, vol. vi. p. 618, 1815). The chyle was collected, by Astley Cooper, from the thoracic ducts of dogs, within three hours of a meal either of animal or vegetable food. It was found that the specific gravity and amount of solid residue differed little on different diets, and the salts present were the same, but the chyle collected after a meal of animal food was much the richer in carbon.

A paper read six months before Marcet's death has been left till the last, because of its exceptional interest. It is entitled "Account of a Singular Variety of Urine, which Turned Black soon after being Discharged" (*Med. Chir. Trans.*, vol. xii. p. 37, 1822), and it presents the earliest description, and a very good one, of a case of alcaptonuria in a male infant of seventeen months. There is a description of the staining of the child's napkins a deep purple colour, which was noticed almost immediately after birth, of the normal tint of the urine when passed and of its blackening on standing. The rapid blackening with alkalis is noted, and the failure of acids to restore the original colour. Only the reducing property of the urine is lacking, for at that date the reduction tests used for the detection of sugars were still unknown. The child was lost sight of and could not be traced, but when, seven years later, Marcet decided to record the case, having no laboratory at the moment, he asked Prout to make some further examination of the long-kept specimens. Prout met with no more success than usually attends the study of the black products of oxidation of aromatic compounds.

The interest of these observations will be obvious when it is pointed out that the discovery of alcaptonuria is ascribed to Bœdeker, who, in 1858, found a second reducing substance in the urine of a glycosuric patient, to which, on account of its behaviour with alkalis, he gave the name of "alkapton," and

which is now called homogentisic acid. The intervening thirty-six years contributed nothing to our knowledge of that remarkable anomaly of protein metabolism.

But that is not all : Marcet goes on to speak of another case which came under his notice twenty years previously, that of a young woman who was subject to daily paroxysms "partaking both of the febrile and hysterical character," during which black urine was passed. "She was likewise subject to a remarkable intermittent affection of the integuments in particular parts of the body, the attacks occurring in irregular paroxysms, and generally following or alternating with the febrile fit. This cutaneous affection usually began with a tingling of the parts, soon succeeded by a considerable swelling or puffiness over an extent of several inches, which lasted for several hours, and ultimately followed by the appearance of a black or dark purple colour, which often continued for some time after the other appearances had subsided. The seat of this affection varied very much, the toes, legs, hips and face being in succession liable to these attacks."

Although there is no suggestion that the colour of the urine was due to blood pigment, and no mention of blueness of the fingers, we can hardly doubt that in this case, which dated from a time when Marcet's experience was comparatively small, was one of paroxysmal hæmoglobinuria with Raynaud's disease. After two or three months the attacks ceased completely, but unfortunately the season of the year is not mentioned. It may be noted that paroxysmal hæmoglobinuria was first described by Dressler in 1854, and by George Harley in 1865 ; that Raynaud described the condition named after him in 1863, and that the association of the two maladies was described by Druitt in 1873.

The *Essay on the Chemical History and Medical Treatment of Calculous Disorders* was published in 1817, and a second edition appeared in 1819. It was translated both into French and German.

In this monograph Marcet brought together the then available knowledge concerning the chemical nature of urinary and other calculi, the analytical methods by which they are most easily distinguished, the geographical distribution of calculous disorders, and the prospects of treatment by other than surgical methods. It was only a short time previously that light had been thrown upon the composition of such stones, first by the discovery of uric acid by Scheele (1776), twenty years later by Wollaston, who recognised four other varieties of urinary calculi, and by the work of Fourcroy and Vauquelin in France, and that

of George Pearson, William Henry and William T. Brande in this country.

In dedicating his book to Wollaston, Marcet pays a generous tribute to the discoveries of that great physician-chemist; but he himself played no small part in the attainment of the knowledge which he sets forth.

He presents a summary of the facts which he has been able to collect, from the very scanty records kept in those days, as to the distribution of calculous disorders in this country and on the Continent, and as to the relative frequency of the several varieties of stones. In pursuit of the desired information he himself analysed many such concretions, and among them no less than 181 specimens from the celebrated Norwich collection, and eighty-seven from that in the museum of Guy's Hospital.

Marcet was the next, after Wollaston, to record cases of cystin calculus, and the first to describe their occurrence in several members of a family. Two previously unknown varieties of urinary calculi are here described, namely the comparatively unimportant fibrinous calculus and the very rare xanthin stone. To the material of this stone Marcet gave the name of xanthin, because of the yellow colour developed when it was acted upon by nitric acid, and his investigations laid the foundation of our knowledge of the previously unknown xanthin bases. Since then five or six other xanthin calculi have been described.

In a chapter which treats of calculi other than urinary, is to be found a clear description of that variety of false intestinal sand which is sometimes passed by people who have eaten freely of pears of certain kinds. The specimen had been submitted to Wollaston, who eventually recognised the grains as being the particles of sclerenchymatous tissue present in the flesh of such pears. Such sand was described more than half a century later—in 1875—by C. Robin, who was obviously unaware of any previous record.

Even at the present day comparatively little additional matter would be needed to bring this monograph up to date, and many of the views expressed in it were far in advance of its day. Marcet recognised fully the renal, as opposed to the vesical origin of urinary calculi, and attributed the deposition of phosphatic layers upon nuclei of other kinds to decomposition of the urine. He was fully alive to the important part played by the animal matter, or cement, which binds together the successive laminae of calculous material, and attributed a share in the growth of calculi to the secretion of ropy mucus from the bladder wall. Lastly, he was under no illusion as to the limited efficacy of the treatment of calculous disorders by administration, or

injection into the bladder, of alkalis or acids, and recognised the fact that such drugs, whilst hindering the deposition of one kind of calculus-forming material, may provoke that of another.

It will be apparent, from this brief review of the writings of Alexander Marcet, that although no great discovery stands to his credit, nor is any wide generalisation associated with his name, almost every paper that he wrote brought forward some new method, or some hitherto unknown facts, not a few of which we are wont to attribute to much more recent investigators. It is clear that he was possessed of the spirit of research, and his contemporaries bore witness to "his laudable zeal in bringing forward the exertions and talents of others in the great field of science and improvement."

There could be no better estimate of Marcet's contributions to knowledge than the following, penned by Nisbet during his lifetime :

"In some of the periodical publications Dr. Marcet appears to great advantage, by the singular merit of his communications, which evince ingenuity, science, accurate experiment and just deductions. They are all of a superior caste, in those volumes too often filled with commonplace and hackened subjects, which owe their merit only to the new form of their dress or arrangement. Dr. Marcet's papers, on the contrary, are marked by originality, precision and clear thinking, impressing unknown or unobserved truths."

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## THE PHYSICAL FITNESS OF MEN ASSESSED BY VARIOUS METHODS.\*

By W. D. HAMBLY, B.Sc., M. S. PEMBREY, M.D., F.R.S., and E. C. WARNER, B.Sc. (From the Physiological Laboratory, Guy's Hospital.)

IN previous papers<sup>1, 2</sup> were given the results obtained in an examination of the physical fitness of male medical students of Guy's Hospital and of women students in the Department of Remedial Exercises, Guy's Hospital, and at Chelsea Physical Training College; the test employed was the reaction of the pulse to exercise. The following conclusions were drawn. The pulse ratio, that is, the ratio between the pulse rate for the two minutes immediately following the given mild muscular exercise and the pulse rate at rest, is a good indication of the physical fitness of the subject. After strenuous exercise, such as running a mile, the pulse ratio is no indication of fitness, the fittest men are those who showed in response to this work the greatest percentage rise in the pulse rate, and the greatest percentage fall to the resting value when the run was finished. The resting rate of the pulse of individuals trained in physical exercises is definitely lower than that of the untrained, but other factors must be taken into account. The average rate of the pulse is lower in the heavier and also in the older subjects, men or women as the case may be. The influence of the time of day appears to be related to the activity and the temperature, both internal and cutaneous, of the subject, and the temperature of the air, as shown by the wet and dry bulb thermometers; the pulse tends to increase in rate with an increase in any of these factors.

The aim of the research, which is the subject of this paper, is the comparison of various tests in use for the assessment of physical fitness. Determinations were made of the following:—pulse ratio, resting pulse, blood pressure, vital capacity, pulmonary ventilation, rate of respiration, holding breath alone or with the mercurial manometer, weight, girth of chest, stem length and total height. The subjects were, with four exceptions, medical students, who were engaged in their pre-clinical studies.

\* Expenses in connection with this research were defrayed by a grant from the Medical Research Council. Mr. Hambly was working on behalf of the Medical Research Council, and Mr. Warner was at the time the University of London Research Student in Physiology.

A classification of their physical fitness based upon personal knowledge and their record of athletic exercise or games was made independently of these tests and the following are the groups :—best athletes 3, very fit men 13, men above the average 19, average men 16, men below the average 9, and unfit men 3, a total of 63 men between the ages of 16 and 39 years. There were four men over thirty, six men nineteen years of age and one sixteen. The observations were made between January and July 1922, and a preliminary account of the results was given at the meeting of the Physiological Society in October 1922. Inquiries on May 27, 1924, showed that all but one of the men were at work at the Hospital at that date; by July 1925 some of the men had qualified and left the Hospital, but all were alive, and the classification appeared to be justified.

The different tests were carried out as far as possible under comparable conditions; no claim is made that the results are rigidly correct, but they represent a careful examination of subjects under conditions which are practicable for trained observers. The tests and their application may be described briefly in the order in which the examination of the subject was made.

1. *Blood Pressure*.—This was taken with the Riva Rocci sphygmomanometer, the armlet being round the arm of the subject sitting in a chair. (a) The systolic blood pressure was determined by the disappearance and appearance of the radial pulse according to digital estimation, the average of the two values being taken as correct. (b) For the diastolic blood pressure the measure was the maximum excursion of the mercury in the manometer as the pressure in the armlet gradually fell, and in most cases also the sensations of maximum pulsation under the armlet felt and indicated by the subject as the pressure in the armlet fell. These two methods gave similar results in all cases.

2. *Pulmonary Ventilation*.—The subject was sitting, and the apparatus consisted of mask and valves, corrugated tubing, a large three-way tap, anæsthetic bag and a wet meter. When the subject is breathing through the mask the tap can discharge the breath either into the outside air or into the anæsthetic bag as may be required. The subject does not watch the indication of the meter but holds the mask tightly over his nose and mouth. The rate of respiration is counted until it reaches a steady value. Any air which may be present is squeezed from the bag and at a given moment expired air is collected for exactly half a minute, the rate of respiration being counted. Then by turning the tap the breath passes into the outside air,



and in this way there is no interruption of the subject's breathing. The air in the bag is measured by passing it through the meter.

The breathing is influenced greatly by conscious or unconscious suggestion and attention was paid to this source of fallacy. A series of measurements was made and results which were obviously doubtful were excluded from the calculation of the average. If the rate was unduly rapid it was counted also when the subject was not wearing the mask and did not suspect that he was being observed or had his attention distracted by the pretence of counting his pulse.

3. *Vital Capacity.*—The subject sitting on a stool about two feet high used the same apparatus as that for the pulmonary ventilation. The deepest possible expiration was taken and was succeeded by the deepest possible inspiration, then the subject applied the mask to his face, breathed into the anæsthetic bag and gave a signal when he could breathe out no more. The tap was turned and the air expired measured through the wet meter. A series of determinations was made and, although the subject did not see the readings of the meter, he was encouraged to increase his output if possible. The highest value was taken to represent the vital capacity. The volumes have not been reduced to 0° C. and 760 mm. Hg., for the application of any such correction gives a false idea of accuracy in the method.

4. *Mercurial Manometer Test.*—The subject sitting in a chair took a deep expiration, followed by a deep inspiration; he then placed and grasped the mouthpiece of the manometer

TABLE

	Number of subjects.	Age in years.	Weight in kilograms.	Height in centims.	Stem length in centims.	Chest in centimetres.		Vital capacity.	
						Measure-ment.	Expan-sion.	Litres found.	Litres calculated.
Best athletes.	3	24 23-24	65.6 62.3-70	173.7 170-180	89.3 86-94	88.5 85-91	5.7 5.7	4.47 3.35-5.3	4.29 4.0-4.9
Very fit men.	13	24 19-36	62.8 51-76.8	170.5 157.5-177	90.2 85-94	87.3 75.5-94	7.0 5-9.9	4.12 2.85-4.85	4.29 3.47-4.7
Men above average.	19	22 19-25	67.1 48-78.4	175.6 161.5-188	91.4 87.5-97.5	87.3 79.5-92	7.8 3.5-11.5	4.26 3-5.35	4.55 3.8-4.8
Average men.	16	24 16-39	60.4 51.4-71.4	174.4 169-181.5	90.5 87.5-95.5	83.8 77-91	7.5 6-13	3.88 2.7-5.2	4.15 3.74-4.7
Men below average.	9	23 19-30	63.8 52.8-84.3	171.6 162.5-190	89.4 83-95	86.1 81-101.5	7.3 5.5-12	4.14 2.5-6.35	4.2 3.62-5.2
Unfit men.	3	21 19-23	58.2 55-73.3	166.1 161.5-172.5	86.8 83.5-92	82.7 74-93.5	5 3.5-8.0	3.62 2.55-4.2	3.92 3.34-4.35

\* Calculated from Dreyer's tables, Class A. Stem  
† Only 18 steps a minute in case of four of the men

firmly between his teeth and lips. A sudden sharp expiration was made to raise the mercury to the level of 40 mm., the stop watch was started, and as soon as the subject was forced to give up the effort it was stopped. A series of determinations was made and the subject was encouraged to the greatest effort. The highest value was taken as the true result. Tricks such as inserting the tongue into the mouthpiece were ruled out, also the subject was told to prevent ballooning of the cheeks, if present, by placing his elbows on the table and pressing his fists against his cheeks.

5. *Holding the Breath.*—No deep breathing was allowed before the test. The deepest possible expiration was taken and was followed by the deepest inspiration, then the breath was held as long as possible, the time being measured by a stop-watch. A series of readings was taken and the subject was encouraged to do his best; the highest value was taken for the record.

In the case of tests (4) and (5) the observation was not always taken in the numerical order, but pauses for rest were made for about four minutes between consecutive efforts. If there was a tendency for air to escape by the nose during the test, the subject either applied a clip or closed his nose with his finger and thumb.

The *Weight*, *Stem length* and *Height* are net values; they were measured when the subject was stripped.

The *Girth of the chest* was taken at the level of the nipples.

The *Pulse* was taken when the subject was sitting at rest,

I.

capacity. Percentage difference.	Pulmonary ventilation. litres per min.	Respira- tion rate per min.	Pulse rate.		Blood pressure in mm. Hg.		Pulse pressure.	Holding breath: seconds.	Mercurial mano- meter test: seconds.	
			Per min.	Ratio.	Systolic.	Diastolic.				
+3.3 -17.2—+15.2			61.7 57—70	2.35 2:24—2:44	122 110—130	95 85—105	27 25—30	75 53—101	60 37—75	Average. Range.
-5.15 -17.8—+9.5	7.6 6.3—10.7	15.6 12—22	67 52—89	2.36 2:10—2:64	118 95—147	93 70—115	25 11—40	87.5 44—125	56 28—87	Average. Range.
+0.9 -11—+12.6	8.27 5.55—13.71	16 7—24	68 55—85	2.59 2:2—3:09	116 100—131	92 75—115	24 15—36	92 50—145	64 47—103	Average. Range.
-5.2 -23—+9.5	8.7 5.8—16	14.6 11—21	73.7 59—88	2.49 2:1—2:82	113 94—137	84 70—98	29 13—57	90 54—137	65 38—142	Average. Range.
-1.9 -21.5—+9.6	6.83 5.47—8.04	15 13—18	75 56—92	2.50—2.63† 2:27—2:79 2:3—2.92†	117 110—130	89 80—110	28 18—37	83 49—133	59 42—80	Average. Range.
-8.8 -23.5—0.0	9.2 7.0—10.9	18 15—24	83 73—91	2.55—2.58† 2:30—2:79	116 114—119	86 85—87	30 27—34	85 49—105	66 0—72	Average. Range.

length and mean chest measurement.

† Only 18 steps a minute in case of one of the men.

and time was allowed for the pulse to slow down after any slight exertion.

The *Pulse ratio* is the value obtained by dividing the pulse rate for the two minutes immediately following the standard exercise by the pulse rate at rest. For the men who were considered unfit or below the average in physical fitness the exercise of three minutes' duration consisted in mounting a box, 18 inches high, 18 times per minute; for all other men the rate was 24 per minute.

The numerous data obtained are not presented in this paper; here will be given in tabular form only the averages and the range of the results for the different groups of men. The disadvantage of the averages, in which the high and the low values cancel out, is counteracted to some extent by the figures for the range. A more extensive series is necessary for a thorough and critical examination; the collection of such data is laborious, but it is hoped that such a physiological survey of medical students may be continued.

The following conclusions are drawn.

*Vital capacity.*—The test is unreliable. The claims made for this test by Dreyer are not supported. The experience of clinicians since the time of John Hutchinson has led to the disuse of this method in the examination of candidates for life insurance. Efficient respiration is not determined by the capacity or mobility of the chest and lungs; the process involves the co-ordination of pulmonary ventilation with the activity of the heart, the circulation and the oxygen capacity of the blood, factors which cannot be determined by the spirometer.

*Mercurial manometer test.*—This is not a test for physical fitness. The highest records are not given by the best men. In some measure it may be a test for sensitiveness to discomfort; obtuse men of poor physique may give excellent results.

*Holding the breath* appears to be no test for physical fitness, but a measure it may be of the sensitiveness of the nervous system. A dull man of poor physique and capacity may tolerate the discomfort far better than an athlete. It might even be argued that quickness of response is one of the safeguards and attributes of a good athlete.

The *pulse rate* at rest and after exercise gives the best indication of physical fitness, especially if the rate of the decline of the pulse after work is determined by counting the pulse in consecutive periods of a quarter of a minute. It is subject to disturbing factors which have been discussed in previous papers, and there is also evidence that in some chronic diseases the heart may be better developed than the skeletal muscles and

other systems of the body. The following table shows the chief results regarding the pulse.

TABLE II.

Group.	Number of men.	Pulse at rest (sitting).			Pulse ratio average.
		Average.	Range.	Rate of 60 or under. No. of men.	
Best athletes . . .	3	61.7	57-70	2	2.35
Very fit men . . .	13	67	52-89	5	2.36
Men above average . .	19	68	55-85	2	2.59
Average men . . .	16	73.7	59-88	1	2.49
Men below average . .	9	75	56-92	1	2.50 for five men. 2.63 for four men with easier exercise.
Unfit men . . .	3	81	73-90	0	2.55 for two men. 2.58 for one man with easier exercise.

For many reasons the pulse affords the best test of physical efficiency.

The heart, as a pump, must be adjusted to the needs of the body; its action will indicate the nervous control and the response to changes of tension in its muscular fibres produced by the filling of its chambers with blood. Adjustment to exercise can be determined readily, but the exercise selected should be a form in daily use by all people, such as walking or going upstairs. The advantage of the latter is that the minimum of work can be calculated correctly from the weight of the subject and the height of the ascent; further, it is possible by altering the number of the steps and the rate of ascent to grade the work done.

There appears to be no real test for physical fitness for every and any kind of occupation other than the old method of trial and error. A man in perfect health may be fit for one form of muscular work rather than another, and his capacity, which is predetermined by his ancestry and development, can be rendered manifest only by the repeated performance of the work in question. The trainers of men, horses, and dogs pick out their subjects by trials, and the employer of labour finds a month on trial the best method for the selection of his workmen.

Physical fitness is relative and does not necessarily mean good physique; the efficient performance of work involves the interaction of all the systems of the body; the condition is so

complex and the power of adjustment so great that no single measurement can be an infallible guide. One man may be physically efficient for a definite piece of work under certain conditions, such as temperature and moisture of the air, which would not suit another healthy man; there is individuality in all men, each has a certain optimum of speed and environment. Any attempt to bring about uniformity must result in failure.

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## THE DIAGNOSIS OF CANCER OF THE STOMACH\*

By ARTHUR F. HURST, M.D., Physician to Guy's Hospital.

FOUR years of work with a diagnostic team have dispelled a good deal of my former pessimism with regard to the possibility of diagnosing cancer of the stomach at a stage in which radical treatment is still possible. We must depend in the first instance upon the recognition by the general public that instead of treating themselves with patent medicines they should at once seek medical advice for digestive symptoms of a kind they have not previously experienced. Secondly, the general practitioner must recognise that, if such symptoms are not the result of some obvious cause and do not rapidly respond to simple treatment, the patient should undergo a complete investigation with the object of settling the diagnosis. I believe that it is possible in a very large majority of cases to decide definitely within ten days of taking a patient into a hospital or clinic whether the likelihood of cancer of the stomach is sufficiently great to warrant operation. The diagnosis can often be established with certainty; in the remaining cases it can be made with so great a measure of probability that an exploratory operation becomes the only justifiable treatment, even if the symptoms are of short duration or slight in degree. It is true that occasionally at operation no organic disease is found, or the disease is of a less serious nature than was anticipated, but this is more than counterbalanced by the fact that it is very rare indeed for the diagnosis to be missed after a complete investigation. If the patient consults his doctor at the right time and his doctor appreciates the value of a complete investigation in suspicious cases, a diagnostic team should be able to obtain sufficient evidence to decide whether an operation is indicated or not within a month of the onset of symptoms.

If a thorough investigation is only carried out in cases in which cancer of the stomach appeared to be the most likely diagnosis, many early cases would escape recognition. Such an investigation should be carried out in all suspected cases of

\* An abstract of this paper formed one of the opening addresses in the discussion on "Cancer of the Stomach" at the Surgical Section of the British Medical Association Meeting in Bath, in July, 1925.

organic abdominal disease before beginning treatment, whether medical or surgical, even if the diagnosis appears to be obvious, and also in cases of impaired general health, loss of weight, loss of strength, anorexia and anæmia, in which a sufficient explanation is not forthcoming.

In the investigation of such a case there are three principal aids to diagnosis—test-meals, radiography, and examination of the stools. A test-meal helps in many cases, the x-rays and examination of the stools in all, but reliable results can only be obtained when the tests are carried out by observers who are familiar with the fallacies of the methods and how they can be avoided.

The statistics I shall refer to have been prepared for me by Dr. N. L. Lloyd from the records of fifty cases investigated at Guy's Hospital (*vide* p. 410), and by Dr. J. F. Venables from a consecutive series of seventeen private cases investigated at New Lodge Clinic.

### (1) *Test-Meal*

A fractional test-meal properly carried out and properly interpreted gives very valuable information in a considerable proportion of cases of cancer of the stomach. The questions to consider are (a) the character of the "resting juice," (b) the acidity during the meal, and (c) the presence of blood.

(a) *The resting-juice.* It is essential that the stomach should be completely evacuated before the meal is given. The "resting-juice" obtained does not often exceed 50 c.c. in normal individuals, though it may be as much as 100 c.c. More than 50 c.c., and certainly more than 100 c.c., suggests the presence of some difficulty in gastric evacuation. The presence of visible food residue or of dissolved starch or sugar in the juice points strongly to organic pyloric obstruction; this can be diagnosed with certainty if the quantity of food is considerable and if much of the meal is still present when the stomach is finally emptied at the end of three and a half hours. If in these cases free hydrochloric acid is present an ulcer is the probable cause; if no free acid is present, and especially if the material removed is of uniformly thick consistence, a growth is almost certainly present; the diagnosis is rendered still more probable if it has a foul odour and contains excess of organic acids.

In the absence of pyloric obstruction information as to the existence of a growth can only occasionally be obtained by examining the resting-juice; Bennett has laid stress on its foul odour in cancer of the stomach, but this was observed in only two of our cases, and Dr. T. W. Turner tells me that it was

present to an equal or greater extent in several of our cases of achlorhydria due to other causes.

Some French observers pay great attention to the microscopical examination of the resting-juice. But Ganz,<sup>1</sup> working in my clinic, found that the epithelial cells and pus cells in the resting-juice were almost invariably present in the same proportion as in the spittle examined at the same time, and the large number of cells seen in cases of achlorhydria was due to the pus and epithelial cells swallowed with the saliva remaining undigested in the stomach. Microscopical examination of the resting contents is therefore only of value if the spittle is examined at the same time ; in some cases of cancer there is great excess of pus cells in the resting-juices in comparison with the spittle; so far as our experience goes this does not occur in chronic gastritis with achlorhydria or in any other condition.

(b) *Acidity.* The value of the fractional test-meal as compared with the old-fashioned one-hour test is shown by the fact that in a consecutive series of 1080 fractional test-meals at New Lodge Clinic complete achlorhydria was found in 14.7 per cent., but free hydrochloric acid was absent in the one-hour fraction in 7.4 per cent. additional cases, which would have been regarded as having achlorhydria if the contents of the stomach at the end of an hour had alone been analysed. The importance of this is well seen in cancer of the stomach, in which the old method showed achlorhydria in 80 per cent. of cases; but with the fractional test-meal free acid was absent throughout in only 63 per cent. of our cases and 50 per cent. of a Mayo Clinic series reported by Hartman.<sup>2</sup> Hartman showed that the acidity was not influenced by the position of the growth, but the longer the history pointing to malignant disease and the more advanced the carcinoma, the more frequently was achlorhydria discovered. On analysing the results of operation five years after gastrectomy had been performed for cancer of the stomach, he found that 44 per cent. of 39 patients who had had complete achlorhydria were still alive, but only 22 per cent. of 41 with free hydrochloric acid.

The presence of free hydrochloric acid cannot therefore be regarded as evidence against the diagnosis of a growth. On the other hand, though complete achlorhydria is present in 4 per cent. of normal young men (Bennett and Ryle) and in 14.7 per cent. of 1080 medical cases admitted to New Lodge Clinic, in which it seemed desirable to give a test-meal, its presence must still be regarded as a point in favour of a growth if the other evidence points in the same direction. It is of special help in the diagnosis from gastric ulcer, as achlorhydria



was only present in one out of a consecutive series of fifty cases at New Lodge Clinic (Chart I).\* The presence of achlorhydria is, however, of no help in diagnosing from Addison's anæmia, in which it occurs in 100 per cent. of cases, or from syphilis of the stomach,<sup>3</sup> in which it is also generally present. Achlorhydria is common in severe chronic gastritis, which is generally secondary to alcoholism or oral sepsis; but whereas in our experience lavage of the stomach before the test-meal is given only rarely leads to the subsequent appearance of free acid in cases of

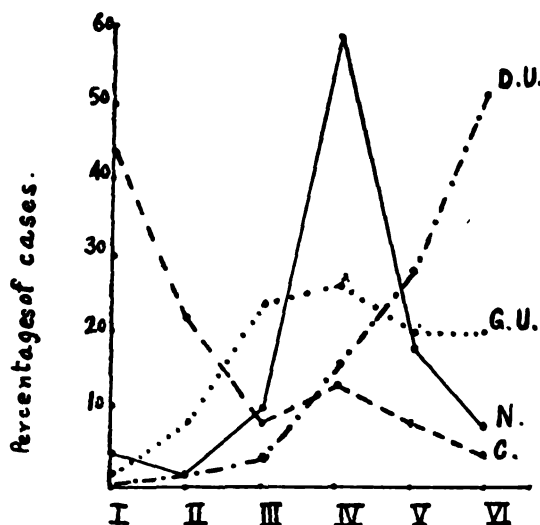


Chart I: Degree of acidity in carcinoma of the stomach compared with the other conditions (Kohiyar).

I-IV, grades of gastric acidity from achlorhydria I to hyperchlorhydria VI (vide J. R. Bell: *Guy's Hosp. Rep.*, lxxii, 302, 1922).

D.U., duodenal ulcer (100 cases).

G.U., gastric ulcer (50 cases).

C., carcinoma of the stomach (15 cases).

N., normal students (100).

growth, it does so in most cases of chronic gastritis as well as in all of chronic gastric ulcer, as in these conditions the achlorhydria is due to the neutralisation of the free acid with the excess of alkaline mucus which is always present. Whenever, therefore, we discover achlorhydria, we invariably repeat the test-meal a few days later after preliminary lavage; if it is still present in a suspected case of chronic ulcer, this should be regarded as probably malignant, and doubt should also be thrown upon a provisional diagnosis of chronic gastritis. In one case in which the symptoms and x-ray appearance suggested

\* Achlorhydria was not present in any of a similar series of 100 cases of duodenal ulcer.

a simple ulcer becoming malignant, and in which achlorhydria was replaced by hydrochlorhydria after lavage, I advised partial gastrectomy: the naked-eye appearance was suggestive of a large chronic ulcer becoming malignant, but microscopical examination showed that it was tuberculous (p. 430).

In the achlorhydria of cancer of the stomach some hydrochloric acid is probably secreted, as the organically combined chloride is often and the inorganic combined chloride is generally greater than normal. This distinguishes it from the large majority of cases of achlorhydria caused by constitutional achylia gastrica, an inborn error of gastric secretion, which is present in about 4 per cent. of normal people and is a predisposing factor of varying importance in a number of diseases, such as Addison's anæmia and subacute combined degeneration (100 per cent.),<sup>4</sup> gallstones (49 per cent.), and chronic appendicitis (33 per cent.) (Bonar.)<sup>5</sup>

(c) *The presence of blood.* In 44 per cent. of our cases obvious blood was present in the resting-juice and in every fraction in sufficient quantity to tinge the whole specimen; in an additional 18 per cent. it was present in one or more fractions. It is rarely present in sufficient quantity to be recognised with the naked eye in gastric or duodenal ulcer unless there has been a recent hæmorrhage, and it is still more rarely seen in other conditions. Its constant presence in association with achlorhydria has only been observed by us in cancer of the stomach. The only case I have seen of Addison's anæmia in which blood was present in the test-meal proved to be secondary to the achlorhydria caused by a growth of the stomach.

## (2) Radiography

In the New Lodge Clinic series the x-rays showed definite evidence of the presence of a growth in every case. In the Guy's series the results were much less satisfactory, definite evidence being only obtained in 50 per cent. of cases. The discrepancy is due to the fact that in the former the examinations were all carried out by Dr. P. J. Briggs, who is an expert in the radiology of the alimentary tract, whereas at Guy's, owing to the pressure of work, the examinations were carried out by various observers, some of whom had had comparatively little experience. With good technique and an experienced observer I think that some abnormality suggestive of growth would be discovered with the x-rays in almost every case. The radiological evidence is, however, not yet quite so trustworthy as in the case of gastric and duodenal ulcer. I am nowadays

unwilling to diagnose an ulcer without *direct* radiological evidence of its presence, but if the other evidence was very strong I would be prepared to advise operation in a case of suspected carcinoma of the stomach, even if nothing abnormal had been seen with the x-rays.

Though a definite deformity can generally be recognised in the outline of the stomach in good radiograms, it is remarkable



FIG. 1.

Radiogram of a stomach showing filling defects produced by a carcinoma of the body of the stomach. The dotted line shows the approximate extent of the tumour. (Dr. P. J. Briggs.)

how normal the appearance may be even with growths of considerable size. For this reason the screen examination is of still greater importance, especially with early growths involving the pyloric half of the stomach. In most cases careful observation will reveal some abnormality in the peristaltic waves. Instead of becoming slowly and steadily deeper as they approach the pylorus, they may disappear entirely, or they disappear and

reappear an inch or more further along the curvature. We have often observed this in the absence of any permanent filling defect which could be recorded on a radiogram. Unfortunately it is in the cardiac half of the stomach, in which peristalsis does not normally occur, that filling defects are most likely to be missed, though this should rarely occur if the patient is examined in various positions, and especially the Trendelenburg position, which results in the complete filling of the fundus with the opaque meal.

Until recently radiographers experienced great difficulty in deciding whether pyloric obstruction was due to cancer or some

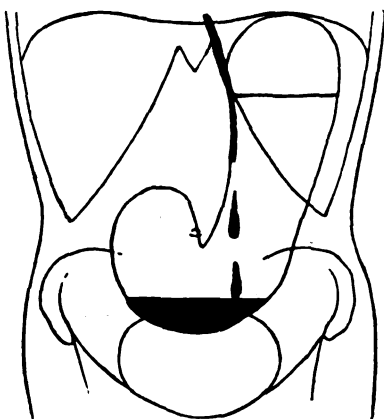


FIG. 2.

First mouthfuls of opaque meal, taken when fasting in the morning, entering stomach already distended with a large quantity of gastric juice as a result of continuous hypersecretion.

other cause. We have found that the difficulty can be overcome by completely emptying the stomach with a Senoran's evacuator before the opaque meal is given. In pyloric obstruction the stomach always contains fluid and often some food residue when the patient is fasting in the morning. Consequently when the opaque meal is swallowed it drops to the bottom of a distended stomach. The transparent gastric contents are generally not observed; a diagnosis of dilatation due to obstruction is made, but as the opaque meal lies in the most dependent part of the stomach the immediate neighbourhood of the obstruction is not clearly seen (Fig. 2). After being evacuated it is found that, though previously distended, the stomach is not permanently dilated, as it nearly always contracts to an almost normal size. Consequently an opaque meal taken now fills the stomach in a

normal manner, and the outline of the pyloric end of the stomach and the progress of the peristaltic waves from their commencement to their end can be satisfactorily investigated (Fig. 3).

I have seen a number of cases in which the most careful abdominal palpation failed to reveal any tumour, but on repeating the examination under the x-ray screen, when the stomach was visualised, it has been possible to recognise a thickening where an irregularity in outline or an abnormality in peristalsis was observed; the method of combined radiography and palpation, which should be carried out by the clinician and not the radiologist because of the necessary exposure of the hands to the rays, may therefore be of great value when neither method alone has led to any conclusive result.

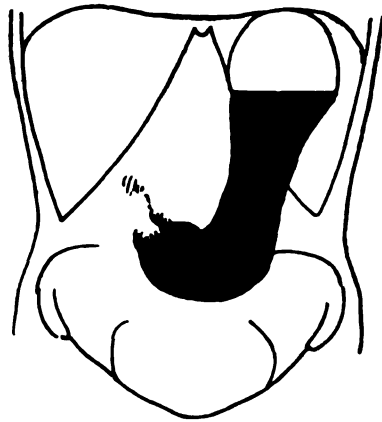


FIG. 3.

Same case as Fig. 2, with opaque meal taken after stomach had been evacuated, so as to show incomplete filling of pyloric vestibule due to growth.

### (3) *Examination of the Stools for Occult Blood*

When blood is swallowed or is derived from an ulcer or growth in the alimentary tract, it is evacuated in the stools partly as acid hæmatin and partly as hæmatoporphyrin. The chemical tests for "occult blood"—traces of blood insufficient to produce any change in the appearance of the fæces—depend upon the conversion of a substance with little or no colour, such as guaiac or benzidine, into a coloured substance when oxidised by hydrogen peroxide in the presence of a carrier, such as hæmatin. Hæmatoporphyrin, which contains no iron, does not give the reaction. The spectroscopic examination of the stools should also be used, as traces of hæmatoporphyrin, which is occasionally present in the absence of acid hæmatin, would

otherwise escape recognition. Moreover, a positive spectroscopic finding is valuable confirmation of a positive chemical reaction, as, although it is much less sensitive, there is less chance of error.

Before examining the stools the patient is given a hæmoglobin-free diet. Chlorophyll should also be excluded, as it gives a feebly positive guaiac reaction, and its many banded spectrum may cause confusion in the spectroscopic examination. A charcoal biscuit is given with the first meal on the restricted diet, and the first and subsequent stools passed when the fæces are no longer blackened by the charcoal are examined. For the guaiac and spectroscopic tests a small amount of fæces is macerated with glacial acetic acid into a thin paste. An equal quantity of ether is then added to extract the pigment; the ethereal extract is poured off, some being kept for the spectroscopic examination. Two or three drops of tincture of guaiac are added to the remainder, a small quantity of ozonic alcohol is then poured in, and a changed colour is looked for at the junction of the two fluids. A "positive reaction" is one in which a deep blue colour rapidly appears; a "feebly positive" reaction is one in which the colour is faint purple, bluish or greenish.

A positive guaiac reaction signifies the presence of occult blood, and a positive spectroscopic examination shows that it is present in fairly considerable quantities. A negative guaiac reaction proves the absence of occult blood, except occasionally at the end of a period of hæmorrhage, when the spectroscopic test may alone be positive, as the traces of blood still present may then be completely converted into hæmatoporphyrin, which gives a characteristic spectrum, but does not give the chemical reaction (Ryffel and Payne).<sup>6</sup>

With the technique described no sign is of more value than the discovery of occult blood in the stools. I have never seen a growth, either of the stomach or the colon, in which occult blood was not present. Thus the guaiac test was positive in 100 per cent. of the Guy's and New Lodge Clinic cases, and a hæmatoporphyrin spectrum was given in 91 per cent. of cases. It is true that occult blood is also present in nearly all cases of active ulcer of the stomach and duodenum, but the x-ray appearance of these two conditions is so characteristic that difficulty in diagnosis rarely occurs.

I am a great believer in the medical treatment of gastric and duodenal ulcer so long as no hour-glass contraction or pyloric obstruction is present. The danger of failing to recognise that a chronic ulcer is becoming malignant is not great, as the

x-rays generally settle the question at once. But in the very earliest stages the appearance may still be that of a chronic ulcer. In such cases the immediate result of treatment does not help, as the symptoms disappear as quickly as in an uncomplicated ulcer. The crater of the ulcer also becomes smaller, though it does not disappear completely as it does in nearly all cases of chronic gastric ulcer. But what is of far more importance is that occult blood persists, however long the treatment continues and however well the patient appears to get. If therefore at the end of a fortnight's treatment symptoms are



FIG. 4.

Lesser curvature ulcer with large crater; stomach filled.

still present, or at the end of a month the crater has not either disappeared or become much smaller, or, most important, if occult blood is present in undiminished quantity, an operation should be advised without further delay. I have only once failed to recognise that an ulcer was becoming malignant, and generally within a fortnight of the commencement of treatment. The one exception is of such importance as demonstrating the value of looking for occult blood compared with all other methods of examination in cases of this kind that I must briefly relate the history of the case.

*Malignant Degeneration of Chronic Gastric Ulcer whilst under observation.*—A man of 66 had had typical symptoms of gastric ulcer for four years. On admission to New Lodge Clinic on February 23, 1924, the diagnosis was confirmed by the discovery with the x-rays of a large crater on the lesser curvature

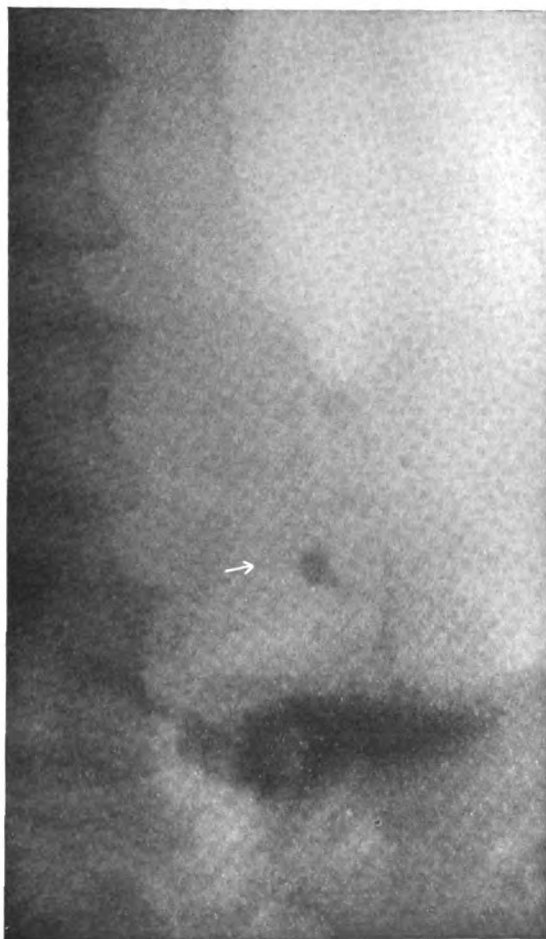


FIG. 5.

Same case as Fig. 4, taken after 6 months' freedom from symptoms; persistent "niche," formed by depressed scar in pancreas following healing of ulcer.

(Fig. 4); this was associated with hyperchlorhydria and the constant presence of occult blood in the stools. Though all symptoms disappeared within a week, I wrote to the patient's doctor when he went home in order to continue treatment after being three weeks in the Clinic that "if occult blood is still present after a few more weeks the question of operation should



be reconsidered, as this is just the type of ulcer which undergoes malignant degeneration." The patient returned for re-examination on April 11 and again on May 18; the x-rays showed that the crater was much smaller, but as occult blood was still present, I advised a continuation of strict treatment. He returned for the third time on June 22. He felt and looked strong and well, had gained five pounds in weight since his first admission and had had no trace of indigestion. At this stage I thought that the small deformity still seen with the x-rays (Fig. 5) must represent the scarred depression in the pancreas produced in the healing of the ulcer. Everything else seemed so satisfactory that I did not feel justified in advising operation simply because a trace of occult blood was still present in the stools.

Early in August, for the first time since the commencement of treatment, he experienced pain; it was quite different in character from the old ulcer pain and was accompanied by nausea and anorexia. No relief followed careful dieting, and he returned to the Clinic on August 31. He had gained another four pounds in weight since June. Nothing abnormal was felt on abdominal examination. Occult blood was still present in the stools, and a most remarkable change had taken place in the x-ray appearance in the nine weeks which had elapsed since the last examination. Instead of a very small niche with a smooth outline there was a large irregular deformity which was unmistakably due to malignant disease (Fig. 6). The hyperchlorhydria was still present, in undiminished degree.

A few days later Mr. L. Bromley performed a partial gastrectomy. A large thick scar was found on the lesser curvature; its centre formed a smooth walled depression in the pancreas. Microscopical examination showed that carcinomatous changes were occurring on one side of the scar, and that the neighbouring mucous membrane was becoming infiltrated. There were no enlarged glands or secondary deposits. In spite of radiotherapy signs of recurrence appeared seven months later.

There can be little doubt that the persistent occult blood was due to the fact that malignant degeneration had already begun in March or April. This sign therefore preceded the earliest symptoms by five months and the earliest radiological changes by at least three months, and no diminution in gastric secretion had occurred in six months. The case also shows how little reliance can be placed on a gain of weight; contrary to what is generally taught, I believe that the majority of patients with cancer of the stomach would gain weight after a fortnight in bed with careful dieting.

(4) *Examination of the Blood*

In every suspected case of cancer of the stomach the blood should be examined. But it is of the greatest importance to

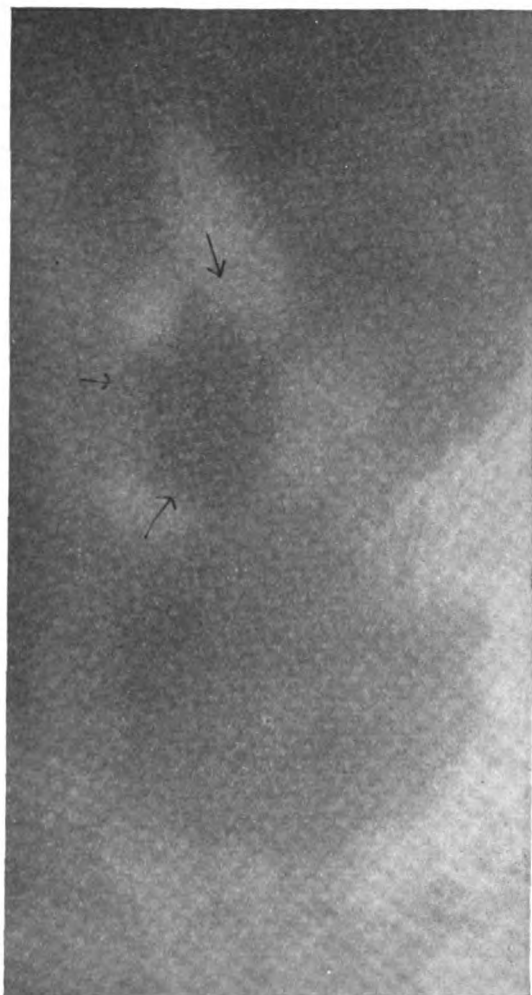


FIG. 6.

Same case as Figs. 4 and 5, taken 6 months after Fig. 4, and 2 months after Fig. 5, showing malignant degeneration of ulcer.

recognise that anæmia is not always present, though it may be severe; in one of our cases the hæmoglobin percentage was only 23. Thus in two advanced cases, in one of which the whole organ was infiltrated to form a leather-bottle stomach, the

hæmoglobin percentage was 95, and in another case the percentage was actually 103.

There should never be the slightest doubt about the diagnosis of Addison's (so-called "pernicious") anæmia from cancer of the stomach. The difficulty in the past has arisen from the character of the blood picture, which the majority of hæmatologists required before making a diagnosis of Addison's anæmia. We now know, largely as a result of Price-Jones' work, that the only constant feature of the blood in Addison's anæmia is megalocytosis. This occurs in no other condition likely to be mistaken for growth; it is present in the very earliest stages of the disease, as for instance in cases of subacute combined degeneration of the cord or Hunterian glossitis, in which no actual anæmia has yet developed, and it also persists after apparent recovery, when the hæmoglobin percentage may be over 100, although in such conditions none of the other features such as the presence of megaloblasts or poikilocytosis are present. Moreover, in Addison's anæmia, though achlorhydria is always present, the stomach is normal in size and outline, and occult blood is never found in the stools; in the active stages, in which difficulty in diagnosis might possibly arise, the indirect van den Bergh's test is positive, though the direct is negative, whereas in cancer both are negative, unless jaundice is present as a result of secondary deposits in the liver, in which case both are positive.

Megalocytosis is never present in uncomplicated cancer of the stomach. I have, however, seen two cases,<sup>4</sup> and others have been described, in which Addison's anæmia occurred as a complication when the cancer had led to complete achlorhydria, so that the essential predisposing conditions for the development of the disease were present. But as the anæmia is secondary to the growth, it only appears at a late stage, in which there is no longer any possibility of radical treatment.

Gastrectomy causes complete achylia; consequently oral sepsis may lead to secondary infection of the intestine and in rare instances to the development of hæmolytic and neurotoxic poisons, which give rise to Addison's anæmia and subacute combined degeneration of the cord. The danger is present to a less extent after partial gastrectomy and even after gastro-enterostomy, if complete neutralisation of the acid which is still secreted occurs, though of course only with total gastrectomy is absolute achylia produced. The danger can be easily prevented if before every gastric operation, whether total or partial gastrectomy, or simply gastro-enterostomy, all oral and nasopharyngeal sepsis is as far as possible eradicated.

The Wassermann reaction should always be tested. In a hundred consecutive patients at New Lodge Clinic, none of whom showed any obvious signs of syphilis or of a disease known to be due to syphilis, a positive reaction was only obtained once (J. F. Venables). It is clear, therefore, that a positive reaction in a case of suspected cancer, at any rate in better-class practice, should be regarded as of considerable significance. One group of cases of syphilis of the stomach is clinically almost indistinguishable from cancer, as the x-rays show a deformity, achlorhydria is present, and occult blood is present in the stools. If in such a case a positive Wassermann reaction is obtained, and the patient appears to be in better general health and has lost less weight than one would expect from the extent of the disease, a trial of active anti-syphilitic treatment should be made, especially if it seems unlikely that the whole mass in the stomach could be completely removed. But if obvious improvement does not occur within two or three weeks, there should be no further delay; I have myself seen three cases of proved cancer of the stomach in patients who gave a positive Wassermann reaction.

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# CANCER OF THE STOMACH

## AN ANALYSIS OF FIFTY CASES AT GUY'S HOSPITAL

By N. L. LLOYD, M.B., Medical Registrar.

THE following analysis of cases of carcinoma of the stomach was undertaken at the suggestion of Dr. A. F. Hurst, with the object of correlating the findings by biochemical and x-ray investigations with the diagnosis, where the latter was confirmed either by laparotomy or by post-mortem examination.

I should like to express my thanks to Dr. J. H. Ryffel for many suggestions in the preparation of this paper, especially as regards the biochemical investigations.

### SELECTION OF CASES

During the years 1921 to 1924 there were 174 cases of carcinoma of the stomach admitted to the medical and surgical wards of Guy's Hospital. In 122 of these cases the diagnosis was confirmed either by operation or by post-mortem examination. The remainder were diagnosed on clinical and other grounds, and since they were considered inoperable they were discharged to receive treatment elsewhere.

Of the 122 cases many were operated on without any investigations being carried out, or were admitted in such a state that investigations were impracticable before they died. There remained fifty cases in which the diagnosis was confirmed and in which sufficient investigations had been carried out for the purpose of this paper. These fifty cases form the basis of the following analysis.

### INVESTIGATIONS

The investigations which have been carried out include a fractional test-meal, examination of the fæces for occult blood, and radiography after an opaque meal. In all the cases a fractional test-meal was performed; x-ray examination was carried out in forty-six cases, and in thirty-eight cases the fæces were examined for the presence of occult blood.

In addition to a summary of the above investigations, a note has been made of the age of the patient, the length of history of gastric trouble, the presence of complications, and

whether the cases were operable or not. The term operable is used here to mean that removal of the growth was performed.

The x-ray examinations were made by the Hospital Radiographers and by the Medical Registrars. The test-meal analyses and examination of the stools for occult blood were carried out by Dr. J. H. Ryffel.

## RESULTS

It must be remembered that these cases are all "hospital" cases, and that for reasons of economy of time and money the investigations have not been as thorough as may be achieved in private practice. Further, it is regrettable that this type of patient has not yet learnt to present himself for examination and advice until symptoms have become really pressing and incapacitating. This is important in considering the number of cases in which removal of the growth was performed.

### 1. TEST-MEALS

#### (a) *Resting Juice*

This was examined in forty-five cases. In thirty-nine there was no free acid. Of the remaining six, four showed normal free acid and two showed hyperacidity.

In twenty-one cases blood was present.

In twelve cases sugar was present from a meal taken twelve hours previously, and in six of these starch was present also. In one case a pint of fluid was withdrawn from the fasting stomach, and in one case barium sulphate was present from an x-ray examination made two days previously.

In only one case were the specimens at all foul-smelling, a condition which Bennett has stated is very common in carcinoma of the stomach.

#### (b) *Acidity*

*Free acid.*—The degrees of acidity were found to be as follows :

Achlorhydria	.	.	.	.	32, or 64 per cent.
Hypochlorhydria	.	.	.	.	8
Normal	.	.	.	.	3
Hyperchlorhydria	.	.	.	.	4
Climbing curve	.	.	.	.	3

Those cases which showed normal or high acid were, with two exceptions, ulcerating growths of the pylorus. The exceptions were an old gastric ulcer become malignant, the position of which was not stated, and a growth two inches from the cardia.

Those cases with a climbing curve were all pyloric lesions.

*Total acid.*—The average normal test-meal shows a difference between the “free” and the “total” acidity corresponding to about 10 c.c.  $\frac{N}{10}$  NaOH per cent. This difference was found to be increased in seven of these cases. In each case the free acid was low, and in addition the emptying time was delayed. Further, in four of the seven cases there was x-ray evidence of involvement of the pylorus as shown by irregularity of outline, and in one other case there was dilatation of the stomach suggesting pyloric involvement. In the sixth case the x-ray appearance was normal. One case was not x-rayed.

### (c) *Emptying Rate*

The normal stomach with a standard test-meal should be empty in from one and half to two and a half hours.

In the present series twenty-three cases showed delay in emptying, seventeen showed no delay, while in ten cases there was rapid emptying.

Of those showing delay, seventeen cases had achlorhydria or hypochlorhydria, two cases showed normal acid, one had hyperchlorhydria, and three gave a climbing curve. With the exception of those with a climbing curve, these cases show degrees of acidity which are roughly in the same proportion as in the total number of cases.

All the cases with a climbing curve showed delay in emptying. This is what may be expected, since the climbing curve is probably best explained on the ground that there is achalasia or spasm of the pylorus or a more permanent partial occlusion, so that there is associated together delay in emptying with absence of alkaline regurgitation. Further, the secretion of gastric juice is continued longer than usual since food is present to act as a stimulus for a longer period.

Rapid emptying was found in ten cases. In all of these there was achlorhydria or hypochlorhydria. In six of these there was obvious bleeding, as shown in the test-meal; an examination of the fæces showed that there was a considerable quantity of blood present in three, while one case only showed a trace of blood in the fæces and none in the test-meal.

### (d) *Bleeding*

Blood was present throughout the test-meal in twenty-three cases. It was present in some specimens in nine cases, and in the remaining eighteen it was absent throughout. In those

cases in which blood was not found there was a considerable quantity of occult blood in the fæces in nine cases, and a trace in the remaining nine.

## 2. BLOOD IN THE FÆCES

The fæces were examined for the presence of blood after the patient had been on a hæmoglobin-free diet for at least three days. The guaiac test was applied, and the spectroscope used to determine the presence of hæmatoporphyrin and acid hæmatin.

Little attention has been paid by the majority of workers to fæcal examinations. The chief reason given by those who do not consider this investigation worth while is that the normal healthy individual may show the presence of traces of blood in the fæces. Bell's investigations, however, show that when proper precautions are taken this is not true.

In Table I the presence of a faint trace of blood is shown by the sign (+), a slightly greater amount by the sign +, and any considerable amount by ++ or ++++. It will be seen that in several cases where there was no blood found in the test-meal there was obvious blood in the fæces, and this additional information is of the greatest value.

In the thirty-eight cases in which the fæces were examined the results were as follows :—

Guaiac	(+)	1
	+	15
	++	19
Hæmatoporphyrin	—	3
	(+)	3
	+	15
Acid hæmatin	++	13
	—	16
	+	5
	+++	14

## 3. ANÆMIA

Examination of the blood was carried out in a few cases. In many it was obvious that the patient was very anæmic. It is usually stated that all patients suffering from gastric carcinoma have some degree of anæmia. It is therefore of interest to note that in two cases there was a normal cell-count and hæmoglobin content. Indeed, in one case the hæmoglobin was 103 per cent.

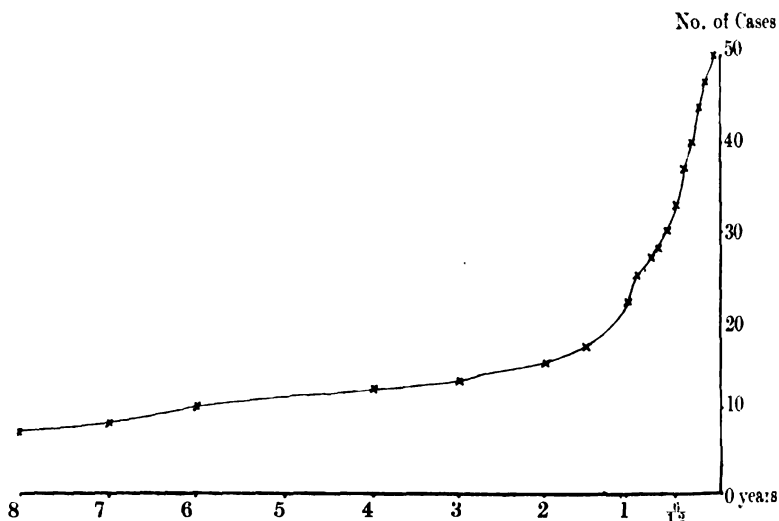
## 4. X-RAY APPEARANCES

Forty-four of these cases were examined by means of the x-rays after an opaque meal. The examination was in every case by screening, and in some cases plates were taken in addition.



In twenty-two cases there was definite evidence of carcinoma as shown by extensive irregularity, "leather-bottle" appearance, annular defect, or persistent deficient filling of the pyloric antrum.

Of the remainder, eight were reported as normal in every respect. In one of the others an old gastro-jejunostomy was present, but the appearance was otherwise normal. In two craters suggestive of gastric ulcer were seen; in five the stomach was dilated with or without delayed evacuation, and in the remainder some abnormality of form or peristalsis was observed.



Showing length of history of gastric trouble. The graph shows at any point the total number of cases whose histories extended back as far as that point or further.

#### LENGTH OF HISTORY

A note has been made in the table of the length of history of gastric trouble of any kind. There were in addition two patients who had suffered from migraine for as long as they could remember.

It will be seen that forty-four per cent. of the patients had had a history of gastric trouble of some kind or another for more than one year, while in fifty-six per cent. the history did not extend further back than one year.

The variation in the length of history is shown in a graph in which the curve represents at any point the number of cases in which the length of history was that number of years or more.

#### RADICAL OPERATION

Removal of the growth was performed in nine cases. In the remainder removal was found inadvisable or impossible owing

to the state of the patient, to the presence of secondary deposits, or to the extent of infiltration of the growth into surrounding tissues.

The following cases were those in which radical operation was performed :—

Case No.	Length of history.	Notes.
4	6 yrs.	Perforated gastric ulcer six months previously, when gastro-jejunostomy was performed. X-ray showed slight deformity of the pyloric antrum, suggesting an ulcer. Test-meal showed some bleeding, with marked regurgitation and low free acid, the stomach emptying rapidly as for gastro-jejunostomy. Faeces contained much blood.
13	2 yrs.	Had had malaria. Wassermann strongly positive. X-ray showed a dilated stomach with active peristalsis, but delay in emptying. Test-meal showed bleeding with hypochlorhydria and definite delay. The faeces contained much blood.
22	33 yrs.	Twenty-five years previously had had hæmatemesis and melæna. X-ray showed a large atonic stomach with no deformity, fair peristalsis but some delay. Test-meal showed normal acidity, bleeding present and considerable delay. The faeces were not examined. There was an ulcer at the pylorus showing colloid carcinoma on section.
28	1 $\frac{6}{12}$ yrs.	X-ray showed stomach normal in size, shape and position, peristalsis active. X-ray six months later showed stomach large, deformity of pyloric end, suggesting carcinoma. Test-meal showed low free acid, rapid emptying and no bleeding. Faeces contained a moderate amount of blood.
30	1 $\frac{7}{2}$ yrs.	X-ray showed irregularity at the pyloric end, tender on pressure. Test-meal showed a large amount of resting juice, containing red cells and pus. There was definite gastric delay, with low acid. No blood except in the resting juice. The faeces contained a moderate amount of blood. Removal was followed by recurrence locally seven months later, when he died.
33	10 yrs.	Had had a gastric ulcer ten years previously. Gastro-jejunostomy. X-ray showed stomach large, gastro-jejunostomy acting freely, no evidence of active ulceration. Test-meal showed high acidity. Faeces showed a small amount of blood present.
36	15 yrs.	For fifteen years had had gall-stones with colic and occasional jaundice. She was not x-rayed. Test-meal showed achylia, no blood. Faeces not examined. Old healed ulcer with malignant changes.
41	6 yrs.	Laparotomy performed six years previously for ? perforated gastric ulcer. Nil found. X-ray showed pyloric stenosis, no evidence of neoplasm. Test-meal showed no free acid, gastric delay but no stenosis. The faeces contained much blood.
45	20 yrs.	X-ray showed small stomach, mobility fair, great deformity of pyloric antrum. Test-meal showed hypochlorhydria, no other abnormality. Faeces contained some altered blood. Laparotomy revealed gall-stones in addition to carcinoma.

It will be seen that there was a history of gastric trouble extending back for more than five years in six out of the nine cases, and that in only one case was there a history of less than one year.\* This case with a history of only seven months died of a local recurrence seven months later.

### APPENDIX OF CASES

1. Male, aged 63. Gnawing pain in the epigastrium for a year, gradually getting worse. Loss of weight and anorexia.

Palpable tumour in epigastrium. X-ray not abnormal; the palpable tumour was above the lesser curvature. Test-meal showed marked hypochlorhydria with some bleeding. The fæces were not examined.

Laparotomy. Inoperable carcinoma of the stomach.

2. Male. Pain in the epigastrium for five months, with vomiting. Sudden hæmatemesis two months before admission.

X-ray showed small defect in pyloric antrum, suggesting early neoplasm. Test-meal showed bleeding, no delay, high acid, suggesting gastric ulcer. Fæces contained much blood.

Laparotomy. Large malignant ulcer on the lesser curvature two inches from the cardia. Secondary deposits in the liver.

3. Male, aged 67. Operation for gall-stones five years previously. For two years had had pain round the heart, which had continued. Loss of weight, vomiting, occasional hæmatemesis.

Palpable tumour. X-ray showed filling defect of pyloric antrum, no delay. Test-meal showed marked hypochlorhydria, with irregular bleeding, and sugar in the resting juice. The fæces were not examined.

Post-mortem. Carcinoma of the pyloric end of the stomach with secondary deposits in the liver.

4. Male, aged 55. Six years indigestion, after walking. No relation to meals. There had been some remissions. Six months before admission had an operation for perforated gastric ulcer, when gastro-jejunostomy was performed. For six weeks had had vomiting with occasional hæmatemesis.

Palpable tumour. X-ray showed slight deformity of the pyloric antrum suggesting ulcer. Test-meal showed marked regurgitation with bleeding, and rapid emptying as for a gastro-jejunostomy. The fæces contained well-marked blood.

Laparotomy. Carcinoma of the pyloric end of the stomach. Partial gastrectomy performed.

5. Male, aged 49. Pain in the epigastrium for six months. Worse for five weeks. Vomiting after meals.

\* On the other hand, in the New Lodge Clinic series of fifteen cases gastrectomy was performed six times, and the history was of less than six months' duration in three of these.

Palpable tumour. X-ray normal; tenderness along lesser curvature. Test-meal showed red cells in the resting juice. No specimen acid to litmus, blood in all specimens, suggesting advanced carcinoma. Hæmoglobin 103 per cent. Red cells 6,000,000.

Fæces contained much blood.

Laparotomy. Inoperable carcinoma of the stomach.

6. Female, aged 60. Continual pain in epigastrium for six months, relieved by vomiting. Much loss of weight.

Palpable tumour. X-ray showed small hypertonic stomach, peristalsis poor, pyloric half very narrow, outline irregular. Test-meal not abnormal. Fæces contained a trace of blood.

Laparotomy. Infiltrating growth of pylorus. Gastro-jejunostomy performed.

7. Male, aged 65. Pain in epigastrium for four years after meals. Loss of weight.

Not x-rayed. Test-meal showed resting juice containing fat globules, delay in emptying, climbing curve with low acid, and blood in the later specimens. The fæces contained altered blood.

Laparotomy. Carcinoma of the pyloric end. Gastro-jejunostomy.

8. Female, aged 42. Pain after meals for eleven months. Relieved by vomiting. Incessant vomiting for six weeks. Loss of weight.

Palpable tumour. X-rayed in horizontal position only. X-ray showed stomach very large and ptosed, mobility fair, meal leaves slowly, considerable deformity in region of pyloric canal. Suggestive of pyloric stenosis, but no evidence of neoplasm. Test-meal showed much solid in all specimens, much gastric delay, pyloric stenosis, traces of bile, and blood present throughout. Free acid present, curve of the climbing type. Blood in fæces.

Laparotomy. Carcinoma of stomach with secondary deposits in mesentery.

9. Female, aged 52. Always suffered from indigestion. Worse for four months. Loss of weight. Anorexia. Vomiting for three months.

Palpable tumour. X-ray showed considerable deformity of pyloric antrum, delay. Tumour corresponded with the deformity. Test-meal showed marked hypochlorhydria, with high total acidity in resting juice, which contained starch, sugar and fat globules. No blood present. Fæces contained altered blood.

Laparotomy. Carcinoma of pyloric end. Gastro-jejunostomy.

10. Male. Pain in left side for eleven months, with anorexia, loss of weight and vomiting.

X-ray showed moderate coloptosis, otherwise not abnormal. Test-meal showed hypochlorhydria, some gastric delay and blood in the last few specimens. Fæces contained altered blood.

Laparotomy. Carcinoma of stomach with secondary deposits.

11. Male, aged 43. Gastritis for seven years. Pain had been different for eight months, and worse for five weeks, with vomiting.

Palpable tumour. X-ray showed vigorous peristalsis, but no food was seen leaving. Test-meal showed hypochlorhydria, with much regurgitation. No blood. Fæces contained a remnant of blood.

Laparotomy. Carcinoma of stomach. Gastro-jejunostomy.

12. Female, aged 61. Indigestion on and off for nine years. Pain worse for two years, after food. Loss of weight. Vomiting.

Visible peristalsis. Not x-rayed. Test-meal showed gastric delay with a climbing curve, bleeding throughout. Fæces contained much blood.

Laparotomy. Carcinoma of stomach. Gastro-jejunostomy.

13. Male, aged 36. Two years indigestion. One year vomiting. Loss of weight. Had had malaria.

Palpable tumour. Visible peristalsis. Wassermann strongly positive. X-ray showed stomach dilated, with active peristalsis, delay in emptying. Test-meal showed definite delay, hypochlorhydria and bleeding throughout. Fæces contained much blood.

Operation. Partial gastrectomy. Growth at pyloric end of stomach.

14. Female, aged 40. Pain in epigastrium eight years previously. Operation (? nature) for gastric ulcer. Recurrence of pain five months before admission. Vomiting.

Palpable tumour. X-ray showed a large ulcer on the lesser curvature, with hour-glass constriction of an organic nature. Test-meal showed highly acid resting juice, containing blood, and gastric delay. Fæces contained much occult blood.

Laparotomy. Carcinoma of the pyloric end of the stomach. Gastro-jejunostomy.

15. Male. Eight weeks indigestion—pain after food and at night. Hæmatemesis and loss of weight.

X-ray showed small hypertonic stomach, which did not dilate. Food passed rapidly to the duodenum. Suggested acute gastritis or leather-bottle stomach. Test-meal showed very low acidity, no delay, and blood in all specimens. Fæces showed much fresh bleeding.

Laparotomy. Diffuse carcinoma of the stomach, inoperable.

16. Male, aged 67. Tenderness in the epigastrium for eleven months, heavy sensation after food, loss of weight. Appetite good.

X-ray showed long hypertonic stomach, peristalsis poor, no irregularity. Duodenal cap not well formed. Test-meal showed high acidity, no delay, no blood. Faeces contained moderate amount of blood.

Laparotomy. Ulcerating growth of pylorus, with enlarged glands. Section of gland showed carcinoma.

17. Female, aged 66. Pain in the epigastrium for five months with vomiting and slight loss of weight.

Palpable tumour. X-ray showed large hypertonic stomach, with irregularity of the pyloric antrum, delay, suggesting carcinoma. Test-meal showed gastric delay, blood in all specimens, hypochlorhydria, with high total acidity, suggesting carcinoma. Faeces contained much altered blood.

Laparotomy. Inoperable carcinoma of the stomach.

18. Male, aged 49. Pain for three months in epigastrium, at irregular intervals. Vomiting after food for a week.

Succussion splash in stomach. Auricular fibrillation. X-ray showed great delay, with deformity of pyloric antrum. Test-meal showed a pint of resting juice, containing blood, starch and sugar. Marked hypochlorhydria, with high total acidity in resting juice, blood in all specimens. Faeces contained much blood.

Post-mortem. Large growth of pylorus.

19. Male, aged 33. Bilioussness (typical migraine) since a boy. Dyspepsia two years previously, which got better. Pain after meals, with vomiting, for a month. Anorexia and loss of weight.

Palpable tumour. X-ray showed leather-bottle stomach. Test-meal showed achylia, rapid emptying, and bleeding throughout. Faeces were not examined.

Laparotomy. Leather-bottle stomach with secondary deposits.

20. Female, aged 49. Gastritis three years previously. Indigestion for four months. Vomiting after meals for six weeks. Loss of weight.

X-ray showed active peristalsis, no irregularity of outline. Test-meal showed low free acid, no delay, no blood. Faeces contained altered blood.

Laparotomy. Carcinoma of the stomach, inoperable.

21. Male, aged 56. Pain ten minutes after food for five weeks. Slight vomiting.

Liver very enlarged and irregular. Ascites. Wassermann strongly positive. X-ray showed deficient filling of the pylorus.

Test-meal showed blood and sugar in the resting juice, no free acid present, bleeding throughout. Fæces not examined.

Post-mortem. Carcinoma of the stomach, with secondary deposits in the liver.

22. Female, aged 64. Chlorosis as a girl. Aged 31 had vomiting attacks for three years. Also had typhoid. Aged 39 had melæna and hæmatemesis. For two months had had pain between the shoulders and round the umbilicus.

Visible peristalsis. X-ray showed large atonic stomach, peristalsis fair, no deformity, considerable delay. Test-meal showed normal acidity, considerable delay, blood present. Fæces contained moderate amount of blood.

Laparotomy. Carcinoma of the stomach. Gastrectomy performed. Ulcer at pylorus showing colloid carcinoma on section.

23. Female, aged 37. Flatulence for seven months. Vomiting for six months.

X-ray showed dilatation and ptosis, with annular defect of the pyloric antrum, suggesting neoplasm. Test-meal showed hypochlorhydria, high total acidity in resting juice, blood present throughout. Fæces were not examined.

Laparotomy. Carcinoma of the stomach, secondary deposits in the liver.

24. Male, aged 40. Progressive indigestion for eighteen months. Pain in the epigastrium and loss of weight.

X-ray showed considerable irregularity of the pylorus. Test-meal showed achylia, sugar in the resting juice, no blood. Fæces showed much altered blood present.

Laparotomy. Large growth involving whole stomach.

25. Female, aged 59. Pain in the epigastrium for six months, after food. Vomiting for two weeks. Slight loss of weight.

Palpable tumour. X-ray showed large filling defect of the pyloric antrum, no delay. Test-meal showed no free acid, sugar in the resting juice, and blood in all specimens. Fæces were not examined.

Laparotomy. Carcinoma of pyloric end of the stomach, inoperable.

26. Male, aged 59. Treated four years previously for gastric neurosis. Since then slight indigestion, vomiting, no loss of weight.

X-rayed three years previously, when report was negative in every respect. Not re-x-rayed. Test-meal showed fairly rapid emptying, almost complete absence of free acid, no blood. Fæces showed a trace of blood only.

Laparotomy. Carcinoma of the cardiac end of the stomach, inoperable.

27. Male, aged 58. Sudden attack of epigastric pain four months previously. Pain lasted for seven weeks. Since then some hæmatemesis. Loss of weight.

Palpable tumour. X-ray showed stomach normal in size, shape and position. Test-meal showed no free acid, no bleeding. Fæces not examined.

Laparotomy. Large growth at cardiac end of stomach. Secondary deposits in pancreas and liver. Section of gland showed spheroidal-celled carcinoma.

28. Male, aged 64. Pain in epigastrium for eighteen months. Loss of weight and hæmatemesis.

Very anæmic. X-ray showed stomach normal in size, shape and position. Peristalsis active. X-ray six months later showed stomach large, deformity of pyloric end suggesting carcinoma. Test-meal showed low free acid, rapid emptying, no blood, suggesting gastric ulcer. Fæces contained altered blood.

Laparotomy. Carcinoma of the stomach. Gastro-jejunostomy performed as a temporary measure. Followed by gastrectomy a few weeks later when his general condition improved. Section showed columnar-celled carcinoma.

29. Male, aged 52. Progressive weakness. Pins and needles in arms and legs for eleven months. Indigestion for nine months. Occasional hæmatemesis. Ataxia for two months. Ulcerated tongue for six months. Loss of weight.

Lemon-yellow colour. Pyorrhœa. Fissured tongue. Spleen and liver enlarged. Sub-acute combined degeneration of the cord. X-ray showed hypertonic stomach, vigorous peristalsis, no tenderness, no irregularity, no delay, no evidence of carcinoma. Test-meal showed achylia, blood present throughout, specimens very foul, suggesting carcinoma of the stomach. Fæces contained much altered blood.

Post-mortem. Extensive carcinoma of the stomach and Addison's anæmia.

30. Male, aged 50. Sudden abdominal pain seven months previously, which disappeared after treatment. For five months frequent vomiting half an hour after meals. Loss of weight. Appetite good.

Palpable tumour. X-ray showed irregularity at pyloric end of stomach, tender on pressure, ? ulcer ? growth. Test-meal showed large quantity of resting juice, containing red cells and pus. Definite gastric delay, low acid, no blood except in the resting juice. Fæces contained much altered blood.

Laparotomy. Carcinoma of the stomach. Gastrectomy. Section showed spheroidal-celled carcinoma. Recurrence seven months later, locally, with death.

31. Male. Abdominal pain for eight months after meals. Loss of weight.



Palpable tumour. X-ray showed large filling defect, with pocket, in upper border of pyloric antrum, suggesting early carcinoma. Test-meal showed blood in the resting juice, normal acidity, slight delay, blood present throughout. Faeces not examined.

Laparotomy. Carcinoma of stomach, inoperable. Section of gland showed papillary carcinoma.

32. Male, aged 35. Pain for three months in epigastrium, gradually progressing, relieved by vomiting. Loss of weight. Anorexia.

Palpable tumour. Emaciated. Sallow. X-ray showed stomach small and deformed, some delay. Test-meal showed pus cells in resting juice, hypochlorhydria, and blood present throughout. Faeces contained much altered blood.

Laparotomy. Carcinoma of the stomach, inoperable.

33. Male. Gastric ulcer ten years previously, for which gastro-jejunostomy was performed. Had had a recurrence of indigestion for some years. Lately vomiting.

X-ray showed stomach large, gastro-jejunostomy acting freely, no evidence of active ulceration, no cause found for symptoms. Test-meal showed high acidity, no blood. Faeces contained a small amount of blood.

Laparotomy. Carcinoma of the stomach. Gastrectomy performed. Section showed columnar-celled carcinoma.

34. Male, aged 50. Pain half an hour after food for three months. Vomiting for two months. Loss of weight.

No tumour palpable under an anæsthetic. X-ray showed large irregular filling defect in greater curvature, typical of carcinoma. Some deformity of the pyloric antrum, slight delay. Test-meal showed hypochlorhydria, gastric delay, bleeding throughout. Faeces contained much altered blood.

Laparotomy. Extensive inoperable carcinoma of the stomach.

35. Female, aged 53. Debility for five months, with dragging pain in the epigastrium, irregular in time. Some vomiting. Loss of weight. Appetite poor.

X-ray showed no abnormality. Test-meal showed no free acid, high total acidity, delay, and bleeding throughout. Faeces contained much altered blood.

Laparotomy. Extensive carcinoma of the stomach, inoperable.

36. Female, aged 64. Migraine since childhood. Gall-stones with jaundice fifteen years before and also six years before. Pain in epigastrium after meals for seven months. Vomiting.

Not x-rayed. Test-meal showed achylia, no bleeding. Faeces not examined.

Laparotomy. Old healed ulcer with malignant change in surrounding tissues. Gastrectomy. Death. Section showed spheroidal-celled carcinoma.

37. Male, aged 59. Indigestion for five months. Vomiting for three months. Loss of weight. Anorexia.

Succession splash. X-ray showed stomach large, peristalsis active, slow emptying, considerable delay. Test-meal showed resting juice of 370 c.c. containing starch, sugar and a trace of blood. Low acidity with marked gastric delay, and slight bleeding throughout. Fæces contained much altered blood.

Laparotomy. Carcinoma of the stomach, inoperable. Secondary deposits.

38. Male, aged 52. Pain under the left costal margin for a year. Frequent vomiting. Loss of weight.

X-ray showed no abnormality. Test-meal showed achylia, rapid emptying, bleeding throughout. Fæces not examined.

Laparotomy. Large inoperable carcinoma of the stomach.

39. Male, aged 65. Loss of appetite, and indigestion for a year. Vomiting for two weeks. Loss of weight.

Palpable tumour. Cachectic. Ascites. X-ray showed stomach dilated, no delay, no other abnormality. Test-meal showed no free acid, slight bleeding. Fæces contained considerable amount of blood.

Post-mortem. Extensive carcinoma of the stomach with secondary deposits in the liver.

40. Male, aged 49. Pain after meals for two months. Vomiting for three weeks. Loss of weight.

Palpable tumour. Liver enlarged. Enlarged glands in supra-clavicular fossa. Not x-rayed. Test-meal showed low acid, rapid emptying, no bleeding. Fæces contained some altered blood.

Post-mortem. Carcinoma of the stomach. Secondary deposits in the liver.

41. Male, aged 53. Six years previously, laparotomy performed for? perforated gastric ulcer. Nil found. Indigestion for two years, no relation to meals. Vomiting for six months. Loss of weight.

X-ray showed pyloric stenosis, no evidence of neoplasm. Test-meal showed no free acid, gastric delay but no stenosis. Fæces contained much altered blood.

Laparotomy. Carcinoma of the pyloric antrum. Gastrectomy. Section showed columnar-celled carcinoma.

42. Male, aged 49. Progressive epigastric pain for three months. Loss of weight.

X-ray showed small stomach, peristalsis active, permanent

## CANCER OF THE STOMACH

Case No.	Fractional Test-Meal.													Feces.			X-ray findings.	Age on admission.	Sex.	Length of history in years.
	Acidity.					Resting juice.				Blood.				Emptying rate.			Guaia.	Hematoporphyrin.	Acid haematin.	
	Free acid.	Blood.	Pus.	Sugar.	Starch.	Achlorhydria.	Hypo.	Normal.	Hyper.	Climbing.	Bile.	Total acid.	Present throughout.	Irregular.	Absent throughout.	Delay.	Rapid emptying.			
1	++	++	++	++	++	++			+		+++	+	++	++		++	+	+	+	1
2	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	5
3	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
4	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
5	++	++	++	++	++	++		+			+++	+	++	++		++	+	+	+	6
6	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
7	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
8	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
9	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
10	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
11	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
12	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
13	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
14	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
15	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
16	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
17	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6
18	++	++	++	++	++	++					+++	+	++	++		++	+	+	+	6

Normal.  
Small defect pyloric antrum.  
Pyloric half not dilated.  
Dilatation. Deformity of pyloric antrum as for ulcer.  
Normal.  
Pyloric half irregular. No peristalsis.  
Marked deformity pyloric antrum.  
Marked deformity pyloric antrum = pyloric stenosis.  
Normal. Vigorous peristalsis.  
Dilated. Active peristalsis. Ulcer: lesser curvature. Hour-glass constriction.  
Leather-bottle stomach. Hypertonic. Peristalsis poor. Irregularity of pyloric antrum. Deformity of pyloric antrum.

[illegible]

partial constriction rather high up. Test-meal showed marked hypochlorhydria, with blood present throughout. Faeces contained altered blood.

Laparotomy. Extensive carcinoma of the stomach adherent to pancreas. Inoperable.

43. Male, aged 55. Pain in the epigastrium, chiefly at night, for four months. Nausea.

X-ray showed marked exaggeration of mobility, rapid emptying, no deformity. Test-meal showed achylia with blood throughout. Faeces contained altered blood.

Laparotomy. Inoperable carcinoma of the stomach.

44. Male, aged 54. Progressive abdominal pain for three weeks. Loss of weight.

X-ray showed hypertonic stomach, mobility much exaggerated, no deformity, slight delay. Test-meal showed achylia, no delay, no blood. Faeces showed altered blood in small amount.

Laparotomy. Large malignant ulcer of the lesser curvature. Inoperable.

45. Female, aged 48. Indigestion since childhood. Severe pain after meals for a year. Loss of weight. Retching.

Palpable tumour. X-ray showed small stomach, mobility fair, great deformity of pyloric antrum and of pyloric canal. Test-meal showed hypochlorhydria, no gastric delay, no blood. Faeces contained altered blood.

Laparotomy. Scirrhus carcinoma of the stomach. Gastrectomy. Also had gall-stones. Cholecystectomy.

46. Female, aged 46. Severe pains in the abdomen eleven years previously. Slight jaundice five months previously. Pain in the right side since.

X-ray showed considerable irregularity of pyloric end of stomach. Test-meal showed hypochlorhydria, fat globules throughout, blood in last specimens. Faeces contained altered blood.

Laparotomy. Carcinoma of pyloric end of stomach. Inoperable.

47. Male, aged 56. Pain in epigastrium for nine months. Loss of weight. Anorexia. Occasional vomiting.

Palpable tumour. Not x-rayed. Test-meal showed no free acid, marked gastric delay, blood present throughout. Faeces not examined.

Laparotomy. Soft growth of pylorus. Inoperable.

48. Male, aged 59. Gastric ulcer fifteen months previously. X-ray at that time did not suggest neoplasm. Pain continued. Loss of weight and vomiting.

X-ray showed irregularity at the pyloric end of the stomach and some tenderness. Test-meal showed complete achylia and delay. The resting juice contained barium from opaque meal of two days previously. The fæces were not examined.

Laparotomy. Extensive carcinoma of the pylorus and lesser curvature. Inoperable.

49. Male, aged 40. Indigestion for twenty years. Pain different for a year. Ascites for a month. Loss of weight.

Palpable tumour. Ascites. X-ray showed narrowing of pyloric half of stomach. Test-meal showed normal acidity, delay, blood in last specimens. Fæces contained altered blood. Hæmoglobin 95%.

Peritonoscopy. Carcinoma of the stomach with secondary deposits in the mesentery.

50. Male, aged 51. Loss of appetite and vomiting for two months. Feeling of fulness an hour after meals. Loss of weight.

Indefinite tumour. Visible peristalsis. X-ray showed nothing abnormal. Test-meal showed acid low and late, delay, blood in the last specimens. Fæces contained altered blood.

Laparotomy. Carcinoma of the stomach with secondary deposits in the liver.

## CHRONIC TUBERCULOUS ULCER OF THE STOMACH

By A. F. HURST, M.D., Physician to Guy's Hospital.

THOUGH a variety of tuberculous lesions of the stomach have been recorded, I have been unable to find any record of a chronic tuberculous ulcer, like that present in the following case. It seems, therefore, to be worthy of detailed description.

A gentleman, aged 46, was admitted into New Lodge Clinic on February 3, 1925, with a ten years' history of epigastric

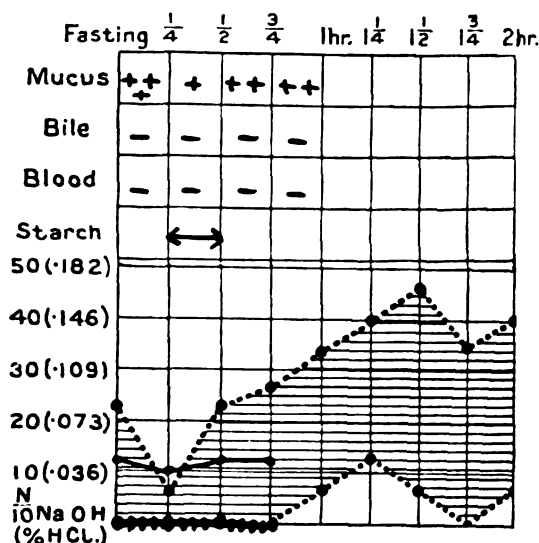


FIG. 1.

Fractional test-meal, showing achlorhydria, excess of mucus, and rapid evacuation.

pain. In September, 1924, he had an attack of profuse hæmatemesis. Since then the pain had become more severe. It began about half an hour after food; it was situated in the epigastrium and bored through to the back. It was accompanied by a sensation of distension, which was relieved by cruetation. A light diet gave relief, but some pain occurred even with starvation. The appetite remained good, and the patient had only lost about 4 lbs. in weight since September,

but he found that he became very easily exhausted and that exertion aggravated the pain.

On examination there was slight tenderness and definite rigidity just to the right of the epigastrium; no tumour was palpable. A test-meal (Fig. 1) showed complete achlorhydria with considerable excess of mucus in the resting juice in each fraction; evacuation was complete in half an hour. On repeating the test-meal after preliminary lavage to remove the mucus, a small quantity of free hydrochloric acid was found to be present (Fig. 2). Every one of twelve stools passed on consecutive days contained occult blood, which varied in amount

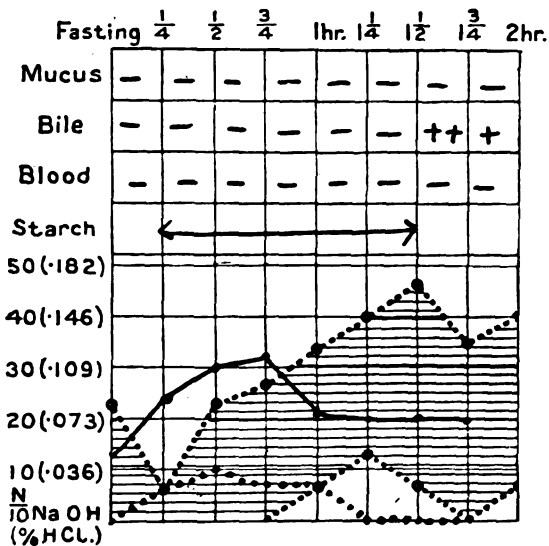


FIG. 2.

Fractional test-meal after preliminary lavage, nine days after that shown in Fig. 1, showing hypochlorhydria, no excess of mucus, and slower evacuation.

from day to day : thus only a faint guaiac reaction was obtained on the 10th, 11th, and 12th, and the spectroscopic examination was negative, but a strong guaiac reaction and hæmatoporphyrin and acid hæmatin spectra were obtained on the 17th. The x-rays showed a stomach of normal size; evacuation was rapid, and the head of the opaque meal had reached the splenic flexure in under four hours. A crater was visible near the centre of the lesser curvature; it was less regular in shape than is usually seen in simple ulcer, the appearance suggesting the presence of malignant degeneration (Fig. 3).

There were no signs or symptoms of phthisis, but the x-ray appearance of the chest suggested the presence of chronic



tuberculous disease; the hilum shadows were excessive and the inner zones of both lungs showed mottling with numerous opacities and linear shadows, but the outer zones and apices were normal. There was no pyrexia.

The Wassermann reaction was negative.

The long history pointed to the presence of a gastric ulcer, but the irregularity of the crater, the variations in the quantity of occult blood in the stools, and the achlorhydria suggested



FIG. 3.

Radiogram showing crater formed by tuberculous gastric ulcer (Dr. P. J. Briggs).

that the ulcer was becoming malignant. Operation was therefore advised.

Mr. R. P. Rowlands operated on February 19. An ulcer,  $\frac{3}{4}$  of an inch in diameter, was found on the lesser curvature, a little below the middle. A partial gastrectomy was performed. The naked-eye appearance was that of a simple ulcer with an unusual amount of infiltration of the surrounding mucous membrane, especially towards the pyloric end, which made the diagnosis of secondary malignant degeneration seem probable.

Sections of the stomach were reported by Dr. F. A. Knott to show a tuberculous process occupying the thickened margins of the ulcer and infiltrating the surrounding regions. The

tubercles were quite typical and occurred freely in the mucosa and submucosa, the wall beneath them being greatly thickened by fibrous tissue. The base of the ulcer was simple fibrous granulation tissue. There was no evidence of malignant change. The diagnosis of tuberculous disease was confirmed by Dr.

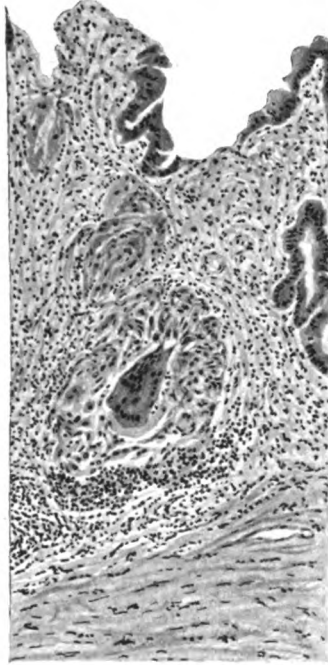


FIG. 4.

Drawing of section through tuberculous ulcer of stomach  
(Dr. G. W. Nicholson).

G. W. Nicholson, to whom I am indebted for the accompanying drawing (Fig. 4).

From the history and morbid anatomy of the ulcer it appears probable that the condition was caused by secondary infection of a chronic gastric ulcer with tubercle bacilli from swallowed sputum. There do not seem to be any criteria which would have made a clinical diagnosis possible from malignant degeneration of a chronic ulcer.

# THE RESULTS OF SPLENECTOMY FOR ACHOLURIC JAUNDICE, ESPECIALLY THE CHANGES IN THE FRAGILITY OF THE RED BLOOD CORPUSCLES

By J. M. H. CAMPBELL, D.M., Beit Memorial Research Fellow,  
and E. C. WARNER, B.Sc.

From the Department of Physiology, Guy's Hospital.

THE object of this paper is to follow the changes after removal of the spleen from patients with acholuric jaundice, especially any changes in the strength of salt solution required to produce hæmolysis of the red blood corpuscles, as the abnormal fragility of these seems to be the most essential change in the disease. The four patients described are Nelly and Albert K., brother and sister, Elsie P., their first cousin, and an unrelated patient, Poppy C., in whom the disease was of the acquired type. The history of these four has already been published in these "Reports,"<sup>21</sup> so here it need only be given very shortly and brought up to date by adding the observations made subsequent to operation.

## *Short History of Patients*

Albert K., born in 1908, was always yellow, though generally less yellow than at birth or during his periodical attacks of shivering and vomiting. He was admitted into Guy's Hospital under Dr. Fawcett in 1920, when the diagnosis was made because of the large spleen, the hæmoglobin percentage of 70, the presence of bile pigment in the plasma but not in the urine, and the hæmolysis of his red cells starting at 0.66 per cent. NaCl. He improved for a time and attended as an out-patient, but little or no progress was made. He had always been small for his age and did not appear to be growing—a complication which is not usual but which has been recorded before.<sup>11</sup> Like some of the earlier patients described,<sup>1, 3</sup> he had large indolent ulcers over the shin-bone which only healed slowly and with difficulty and broke down again. His health was far from satisfactory and it seemed that unless something more could be done to help him he would be unable to earn his living, especially as his hæmoglobin was often as low as 50 per cent.

He was re-admitted and his spleen was removed by Sir Alfred Fripp in November 1921. Cultivations from the spleen, which showed a few adhesions and was eight inches in length, remained sterile. After the operation there was a rapid change in the fragility of his red cells (see later and Table I) and the hæmoglobin percentage rose to 78, but even at the beginning of 1923 the improvement in his general condition was not what had been expected. However, by October, two years after the operation, he had grown considerably and had improved so much that his sister and cousin, with whom the possibility of an operation had been discussed but left in abeyance, suggested that they should undergo the same treatment. In 1925, four years after the removal of his spleen, he was working hard and feeling very well, though still thin and rather liable to colds. His hæmoglobin percentage was 95 and there had been no return of the yellow colour. The fragility was still abnormal, starting at 0.6 per cent. NaCl, though less so than before his operation, when it started at 0.66 per cent. NaCl.

His elder sister Nelly, born in 1896, had a much more severe form of the disease. She was first admitted to Guy's Hospital in 1916 and again in 1920 under Dr. Fawcett, when acholuric jaundice was diagnosed. Her spleen then reached six inches below the costal margin and her hæmoglobin was only 50 per cent. She improved with iron and arsenic during a subsequent pregnancy which had no complications, though the enormous spleen gave her great discomfort. Her blood at that time was partially hæmolyzed at 0.6 per cent. NaCl and almost completely at 0.45. Largely because of the much greater discomfort and disability of her second pregnancy, and because in October 1923 she appeared in such bad condition compared with her brother, she decided that it was worth while having an operation.

In January 1924 Mr. R. P. Rowlands removed the spleen and also the gall bladder, as the latter contained a large number of small pigment stones; during one of her attacks of vomiting and pyrexia her urine had contained bile pigment, but her fæces then appeared to contain the usual amount of pigment and she had never had any symptom pointing to gall-stone colic. The spleen weighed just over 1000 grms., and certain comparisons were made between the blood from the splenic and systemic veins (see later).

For the first four or five days after the operation and again at the beginning of the third week there was a slight degree of pyrexia with some pain referred to the left shoulder, from which she soon recovered, and was then sent to a convalescent home. Two months later she still did not look at all well, was very

thin and again complained of pain in the left shoulder; Elliott<sup>16</sup> has recorded similar signs and suggested that they are due to the rupture of adhesions between the spleen and diaphragm with some damage to the latter. After this date she began to improve and in 1925 she appeared in perfect health and was delighted with the result of her operation. The fragility of her red cells was still abnormal though less so than before the operation (see Table I).

Elsie P., first cousin of these two patients, who was born in 1895, had also a severe form of the disease. She was diagnosed as a case of acholuric jaundice in 1914 by Dr. Robert Hutchison when she was a patient in the London Hospital; her hæmoglobin was then as low as 38 per cent., but it had been even lower (15 per cent.) during a previous admission. In 1923 she still had periodical attacks, when her colour became deeper, and as she was frequently incapacitated from her ordinary household work she was admitted into hospital, and Mr. Rowlands removed her spleen in November of that year; it weighed 980 grms., and sections showed that there was some fibrosis of the pulp with atrophy of its cells, dilatation of the capillaries and atrophy of the Malpighian corpuscles. She made a good recovery from the operation and in 1925 was in good health except for "indigestion." She was not anæmic and had quite lost her yellow colour, but the fragility of her red cells, which had improved a little six months after the operation, had returned nearly to its previous condition.

The fourth patient, Poppy C., had "acquired" acholuric jaundice and her history has already been given.<sup>21</sup> Splenectomy was performed by Sir Alfred Fripp in November 1919. Her case was rather anomalous, as she was admitted to hospital for extreme menorrhagia in addition to the usual signs of acholuric jaundice. Although originally a local cause for the menorrhagia had been sought and not found, the uterus was removed in 1924 by another surgeon because of her persistent anæmia and the continued menorrhagia in spite of the splenectomy. She was then quite free from jaundice and the fragility of her red cells had improved but was not normal. Her hæmoglobin was now low because of the persistent bleeding. A year later she was again admitted after a small hæmatemesis, but unfortunately discharged herself without any investigations or treatment.

#### *Changes after Splenectomy*

*The hæmoglobin percentage and the red and white corpuscles.*—In the three familial cases the percentage of hæmoglobin and

TABLE I.  
BLOOD COUNTS, AMOUNT OF CHOLESTEROL IN THE BLOOD AND FRAGILITY OF THE RED CELLS WITH HYPOTONIC SALINE IN ALBERT K., ELSIE P., NELLY K. AND POPPY C. BEFORE AND AFTER SPLENECTOMY.

	Date.	Red cells. (millions).	Hæmoglobin (percentage).	Colour index.	White cells.	Cholesterol (mgms. per 100 c.c.).	Fragility of red cells with solutions of NaCl:					
							0.42	0.45	0.48	0.54	0.60	0.66
Albert K.	Nov. 16, 1920	4.8	69	0.75	9,000	—	+++	+++	++	++	+	(+)
"	Nov. 25, 1921	4.8	50	0.5	14,000	—	+++	+++	++	++	+	(+)
"	Nov. 25, 1921	5.1	78	0.77	17,000	Splenectomy	+	+	+	+	(+)	—
"	Dec. 6, 1921	5.0	78	0.78	18,000	—	+++	+++	++	++	—	—
"	Dec. 20, 1922	—	82	—	—	—	+++	+++	++	++	—	—
"	Feb. 1922	—	84	—	—	—	+++	+++	++	++	—	—
"	May 1922	—	93	—	—	—	+++	+++	++	++	((+))	—
"	July 1925	—	—	—	—	—	+++	+++	++	++	—	—
Elsie P.	Oct. 1914*	2.3	35	0.76	7,000	—	+++	+++	++	++	(+)	•
"	Sept. 1923	3.0	51	0.85	11,000	—	+++	+++	++	++	(+)	—
"	Nov. 7, 1923	—	—	—	—	100	+++	+++	++	++	(+)	—
"	Nov. 9, 1923	—	—	—	—	Splenectomy	+	+	+	+	+	(+)
"	Nov. 11, 1923	4.1	73	0.80	17,000	—	+++	+++	++	++	+	(+)
"	Nov. 11, 1923	3.9	74	0.94	17,000	—	+++	+++	++	++	+	(+)
"	Nov. 20, 1923	4.7	83	0.83	18,000	160	+++	+++	++	++	—	—
"	Dec. 19, 1923	5.2	69	0.68	14,000	164	+++	+++	++	++	—	—
"	Feb. 1924	5.7	88	0.77	16,000	160	+++	+++	++	++	((+))	—
"	May 1924	—	—	—	—	—	+++	+++	++	++	—	—
"	July 1925	—	—	—	—	—	+++	+++	++	++	((+))	—
Nelly K.	Sept. 1916	4.6	78	0.8	12,000	—	+++	+++	++	++	+	(+)
"	Dec. 1920	1.8	38	1.0	10,000	—	+++	+++	++	++	+	(+)
"	May 1921	3.5	62	0.9	—	—	+++	+++	++	++	+	(+)
"	May 1922	3.4	58	0.9	—	—	+++	+++	++	++	+	(+)
"	Jan. 11, 1924	—	63	—	9,500	83	+++	+++	++	++	+	(+)
"	Jan. 24, 1924	—	76	—	—	78	+++	+++	++	++	+	(+)
"	Jan. 24, 1924	—	—	—	—	Splenectomy	+	+	+	+	+	(+)
"	Feb. 1924	—	—	—	—	73	+++	+++	++	++	+	(+)
"	March 21, 1924	—	76	—	—	132	+++	+++	++	++	+	(+)
"	July 1924	—	82	—	—	170	+++	+++	++	++	+	(+)
"	March 1925	—	92	—	—	—	+++	+++	++	++	+	((+))
"	July 1925	—	—	—	—	—	+++	+++	++	++	+	(+)
Poppy C.	Nov. 1919*	4.2	99	1.1	9,000	—	+++	+++	++	++	(+)	•
"	Nov. 17, 1919	—	—	—	—	Splenectomy	+	+	+	+	+	(+)
"	Nov. 1920	4.9	82	0.84	26,000	—	+++	+++	++	++	(+)	—
"	May 1921	4.7	90	0.55	—	—	+++	+++	++	++	(+)	—
"	March 1924	—	—	—	—	146	+++	+++	++	++	—	—

• Denotes two determinations which were not done by the authors.

Partial hæmolysis nearly complete more than 85 per cent., generally more than 95 per cent.  
 Partial hæmolysis 50-85, generally 50-75 per cent.  
 Partial hæmolysis 25-50 per cent.  
 Slight hæmolysis less than 25 per cent.  
 Very slight hæmolysis, about 5 per cent.

the number of red cells rose almost immediately after operation, and in the acquired case there was not much change, as the percentage was high already. This is as would be expected, for even in health and still more in acholuric jaundice the spleen is the site of the destruction of red cells. But in this particular the patients with acholuric jaundice contrast with normal animals in which, according to Pearce,<sup>17</sup> the percentage of haemoglobin falls after splenectomy, starting at once and reaching its minimum value in five or six weeks and returning to normal some time between three and ten months. Hitzrot has produced evidence of similar changes in man.<sup>19</sup> Pearce believes that this temporary fall in haemoglobin is not merely due to the loss of blood, because it is more than is found after other major abdominal operations, and continues for two or three weeks. It is, however, doubtful if he has taken sufficient account of the large store of blood which can be retained in the spleen, and of the possibility that where this is not lost it may gradually be added to the blood as it is needed, in the period of recovery after operation, just as Barcroft has found to be the case during exercise.<sup>20</sup> The changes in the red cells and in the percentage of haemoglobin seem to go together. The changes in the white cells were followed just after the operation, and in each case they were more numerous then, generally about 20,000 per cub. mm., and in Elsie P. this change continued for six months. In normal men and animals removal of the spleen induces an increase in the number of leucocytes, which at once rises to about 30,000 or more, but soon drops to 20,000, from which figure it returns more slowly to the normal, which it reaches about the fourth month. In this respect it is reasonable that patients with acholuric jaundice should behave in a similar way, as there is no evidence that the leucocytes are affected in the disease.

*Changes in the cholesterol content of the blood after splenectomy.*—Eppinger<sup>10</sup> found that in dogs there was an increase of fat and cholesterol in the blood after removal of the spleen—the effect reaching its maximum in two weeks and lasting for at least two months. This was confirmed by King<sup>12</sup> and more recently by MacAdam and Shiskin,<sup>26</sup> who found that in three cases of acholuric jaundice and in one of splenic anaemia the amount of cholesterol in the blood was increased after splenectomy.

In two patients we have found the same thing and the figures are given in Table I. Taking the average of these six results, the amount of cholesterol in the blood increases rapidly at first and then more slowly, reaching the normal in about three

months. The average figures were before operation 105, two weeks after 122, four weeks after 146, eight weeks after 173, and twelve weeks after 186 mgrms. of cholesterol per 100 cub. cm. plasma. At first this change was thought to be of importance in connection with the decreased resistance of the red cells to hæmolysis and the improvement in this condition after splenectomy, because of the well-known property which cholesterol possesses of protecting the cells against hæmolysis by saponin. This question has recently been discussed by one of us,<sup>30</sup> and it is clear that the actual quantity of cholesterol cannot be an index to the fragility of the red corpuscles, because in a large number of pathological conditions the two do not vary together. It seems more probable that it is the ratio of cholesterol to lecithin which is significant, and this will be further considered in discussing certain differences found between blood from the systemic and splenic veins.

We should like to take this opportunity of drawing attention to a mistake in a paper by one of us,<sup>30</sup> where it was stated that in one case of acholuric jaundice the resistance of the red cells against hæmolysis by saponin was found to be less than normal. This has not been confirmed in five patients examined since; in two the resistance was slightly less, in two it was slightly more than in the normal control, and in the fifth it was the same, so that there was no significant difference. In the usual method of estimating hæmolysis by NaCl the hæmolysis is sufficiently complete for readings to be taken as soon as the corpuscles have settled—say in two hours—but with saponin, with the very varying concentrations which were used, hæmolysis is obvious in some tubes at once and in others appears more slowly, so that sometimes final readings cannot be taken for twenty-four hours. Probably the error must have arisen from neglect of this factor of time; Meulengracht has found the resistance of the red cells against hæmolysis by saponin normal and has quoted similar results by two other workers.<sup>23</sup>

*Changes in fragility of the red blood corpuscles after splenectomy.*—As these changes are important for understanding the pathology of the disease they have been followed closely and are given in full in Table I. The method we have used is that described by Vaquez and Ribierre,<sup>2, 4</sup> and the red cells have been washed with isotonic saline as suggested by Widal.<sup>6</sup> The blood has been drawn from the basilic vein of the arm and, to prevent clotting, mixed as soon as possible with sufficient potassium oxalate to make the solution 0.5 per cent. The blood was then centrifuged and, after the plasma had been removed,



TABLE II.  
QUANTITATIVE ESTIMATION OF PERCENTAGE OF CORPUSCLES HEMOLYSED BY VARIOUS STRENGTHS OF NaCl (WASHED CORPUSCLES).

Subject.	Date.	Percentage haemoglobin.	Percentage of corpuscles haemolysed at the following strengths of NaCl Solution :												
			0.0	0.36	0.39	0.42	0.45	0.48	0.54	0.60	0.66	0.72	0.78	0.90	
Normal control.			100	77	14	(tr)									
Nelly K.	Jan. 11, 1924	{ 63	100	100	100	100	100	90	50	40	17	1 } (7)			
"	Jan. 24, 1924		{ 76	100	100	100	100	80	67	63	52		15		
"	Feb. 2, 1924	{ —	{ (100)	(100)	(100)	(100)	(92)	(75)	(60)	(55)	(26)				
"	March 21, 1924		100	100	100	100	97	90	62	22	8	(tr)			
"	July 1925		{ 76	100	100	100	100	100	70	38	8				
		{ —	100	100	80	80	61	50	24	6					
Elsie P.	Feb. 1924	{ 60	100	100	100	80	50	27	6						
"	July 1925		{ (88)	100	100	91	91	57	30	6	(tr)				
Albert K.	July 1925	{ 95	100	100	100	100	66	33	11	3					

• The figures in brackets are not comparable with the others, as they were obtained on corpuscles from blood from the splenic vein (see text).

washed with isotonic saline and centrifuged twice more. The corpuscles (0.2 c.cm.) were then mixed with 2 c.cm. of saline of various strengths and the degree of hæmolysis was recorded as soon as sufficient sedimentation of the red corpuscles had taken place. Generally the strength of the saline solutions ranged from 0.36 to 0.66 and the amount of hæmolysis was recorded as nearly complete, partial (++), partial (+), slight or very slight. This reading was always made by one of us (J. M. H. C.) in order that the results should be as closely comparable as possible, and from time to time they were compared with a normal control. But after some time one cannot be certain that equal amounts of hæmolysis are being recorded by the same signs, with a qualitative method of this sort.

In all the later determinations the amount of hæmolysis was measured quantitatively over a wider range from distilled water to isotonic saline by a technique evolved by Mr. A. C. Hampson. All these results, which were obtained by E. C. W., are given in Table II. Chauffard's original results were expressed in a somewhat similar way,<sup>5</sup> and Meulengracht has also worked out the percentage of corpuscles hæmolysed by the various strengths of saline.<sup>23</sup> The method consists in filling the graduated tube of the Haldane hæmoglobinometer to the 20 mark with the solution of hæmoglobin obtained from the tube of corpuscles in distilled water. The hæmoglobin to be measured is withdrawn from above the settled corpuscles in the 0.36 per cent. saline tube and placed in an exactly similar hæmoglobino-meter tube, and water is added to the first hæmoglobin solution until the colours match. The reading is again taken in the first tube. Water is then further added until a match is obtained with the hæmoglobin in solution in tubes 0.39, 0.42, 0.45, etc. in turn; from these readings the percentage of completely hæmolysed corpuscles in tubes 0.36, 0.39, 0.42 may be compared with that in the tube of distilled water. From the occasions when the readings have been done in both ways they may be compared, and "nearly complete" was generally above 90 per cent., "partial (++)" generally from 50 to 80 per cent., "partial (+)" from 25 to 50 per cent., and "slight" or "trace of hæmolysis" less than this (see Tables I and II).

The change produced soon after splenectomy was quite noticeable. In one patient (Elsie P.) where the blood was examined two and four days after the operation the resistance to hæmolysis was diminished, but in each case where the blood was examined at a later period after the operation (generally about a month) the resistance was increased. The usual result seems to be that for about a year after operation the fragility

is becoming less abnormal, but after that there is no further improvement, or may even be some return towards the condition before splenectomy. This was noticed after about  $3\frac{1}{2}$  years in one patient and after about eighteen months in the other two with familial form of the disease, but not noticed after four years in the one "acquired" case. No great stress can be laid on this, because it is difficult to be sure that the standard between slight and very slight hæmolysis, etc. remains the same after a year, but there is also one quantitative determination on Elsie P. (see Table II). Possibly these relapses are due to compensatory hypertrophy of hæmo-lymph or other glands in the absence of the spleen, but they are not accompanied by any clinical changes.

What we do wish to emphasise is that in these four patients, nineteen determinations of the fragility after splenectomy never gave a result which could be considered normal. There has always been some hæmolysis with 0.54 per cent. NaCl and a considerable amount with 0.48 per cent. NaCl (with the exception of Elsie P. in May 1924, always more than 25 per cent.); on the other hand, in the normal people whom we have examined we have rarely found any hæmolysis with 0.48 and only occasionally a little with 0.45 per cent. NaCl. Evidently for at least three or four years after splenectomy, in cases where the clinical result is excellent, the blood remains typical of acholuric jaundice, though the fragility of the red cells is of a less severe degree than before.

#### *Observations by Others on the Effect of Splenectomy*

Various observations have now been published showing the effect of splenectomy on the fragility of the red corpuscles. At one extreme Dawson has published the well-known case of acholuric jaundice of Spencer Wells, where the fragility was still abnormal after twenty-seven years,<sup>14</sup> and Thursfield has published one where it was said to have returned to normal.<sup>15</sup> The former observation represents the more usual finding. Two larger collections of results are those of Giffin<sup>18</sup> and Meulengracht.<sup>23</sup> Giffin had followed eight cases, and though one had become normal, the average improvement was not very great. Most of his observations were made one or two months after operation, but one was after twenty months. This last patient still showed greatly increased fragility, but it had not been determined before operation, so there may have been some improvement. Unfortunately Giffin only recorded the point at which hæmolysis was complete, and this is less characteristic than the point at which hæmolysis commences. Before opera-

tion the average was 0.42 and the range 0.40 to 0.47; after operation the average was 0.40 and the range 0.32 to 0.46, or, excluding one case, 0.38 to 0.46; the normal average being 0.36 and the range 0.32 to 0.39. Shortly, the improvement was only one-quarter of what was needed to become normal with the one exception already referred to.

Meulengracht has followed nine patients generally for from two to six months after operation, and in two instances for two and a half years. He has recorded the points of commencing, middle and complete hæmolysis and examined a large number of normal subjects to obtain a good index of the range of natural variations. These results are given in Table III, which shows that the point at which hæmolysis commences shows a much more striking change than the point at which it is complete, which should therefore always be measured; that in every case of acholuric jaundice hæmolysis commences at a point well beyond the normal range; that there is much less change in the point at which hæmolysis is complete; and that after splenectomy there is some improvement in the fragility, but that it does not return to normal. In the two patients traced after two and a half years the point of commencing hæmolysis had been reduced from 0.7 and 0.58 to 0.56 and 0.54 respectively. Meulengracht's figures are not directly comparable with ours because they were obtained with unwashed and ours with washed corpuscles; as regards complete hæmolysis they do not show very close agreement with Giffin's for the same reason, though both give almost the same normal range. The washed corpuscles are hæmolysed rather more readily, and this is probably balanced by the fact that Meulengracht determined the amount of hæmolysis quantitatively, while with naked-eye colour comparisons a tube in which hæmolysis is nearly complete looks the same colour as one in which it is complete.

TABLE III.

RANGE OF HÆMOLYSIS IN NORMAL SUBJECTS AND THOSE WITH ACHOLURIC JAUNDICE BEFORE AND AFTER SPLENECTOMY (MEULENGRACHT).

Condition.	Strength of NaCl solution producing		
	Commencing hæmolysis.	Middle hæmolysis.	Complete hæmolysis.
Normal (20 subjects)	0.42-0.46 (0.48)*	0.38-0.40 (0.42)*	0.32-0.36 (0.38)*
Acholuric jaundice (28)	0.58-0.78	0.42-0.60	0.32-0.42
Acholuric jaundice after splenectomy (9)	0.52-0.62	0.44-0.52	0.30-0.38

\* Indicates a single result which was just outside the usual range.

Our results agree with these two observers and with those of Dawson,<sup>14</sup> Roth,<sup>8</sup> Kahn<sup>9</sup> and others, in showing that in the great majority of cases the fragility of the red blood corpuscles has not become normal a considerable time after splenectomy, in spite of the disappearance of the clinical features of the disease.

*Differences in the Blood from Systemic and from Splenic Veins*

At the operation on Nelly K. blood was taken from the splenic vein and compared with blood taken simultaneously from a vein of the arm. Of course there was some stagnation in the spleen from the time it was clamped till it was removed, but this was reduced to a minimum. Both samples showed complete hæmolysis up to 0.42, but at each strength tested from 0.45 to 0.72, on the average 6 per cent. more corpuscles were hæmolysed in the splenic blood (see Table II). No significant difference was found between the cholesterol content of the splenic and systemic blood, but there is some evidence that the spleen removes cholesterol from the surface layers of the corpuscles (as well as from the plasma), a change which might be responsible for making the cells more readily hæmolysed.

TABLE IV.

DIFFERENCE OF HÆMOLYSIS OF RED CORPUSCLES BEFORE AND AFTER THE  
PASSAGE THROUGH THE SPLEEN (BOLT AND HEERES).

Strength of sodium chloride.	Percentage of corpuscles hæmolysed	
	In carotid blood.	In splenic blood.
0.81	0	0
0.72	0	5*
0.63	0	30*
0.54	20*	60*
0.45	50*	85
0.36	75	100
0.27	100	100

Strisower and Goldschmidt<sup>13</sup> and Eppinger<sup>20</sup> found that the red corpuscles from the splenic vein underwent hæmolysis more readily than those from the systemic blood. Bolt and Heeres<sup>34</sup> confirmed this by some very careful work on sheep's blood and found big differences (see Table IV, which gives the average of twenty determinations), in every case the blood

\* These figures look high and must not be compared with others in this paper, as they refer to sheep's blood.

from the splenic vein undergoing hæmolysis more readily. The only criticism which can possibly be levelled against their results is that the blood from the carotid artery was defibrinated at once, while the spleen had to be dissected out with the minimum of injury to the vessels and corpuscles before the blood was obtained. They do not state how long this took, but recognise that the stagnation in the spleen may have magnified the difference, though they point out quite rightly that stagnation in the spleen does occur under certain physiological conditions. Stagnation in the kidney produced no such effect.

The other important discovery made by these observers was that the change which was produced by the spleen was in the outer layers of the corpuscles, because if they were washed with a modified Ringer's solution the red cells from the spleen reacted similarly to those from other parts of the body.

Accepting the work of Brinkman and Van Dam,<sup>22</sup> that diminished resistance of the red cells implies a decrease in the proportion of cholesterol to lecithin in the surface layers of the corpuscle, the spleen may have removed cholesterol or added lecithin. Bolt and Heeres think that added lecithin is the most important change, and if this is so the washing with "saline" presumably removes some of the lecithin. As under ordinary conditions washing the corpuscles makes them less resistant, and as it is difficult to imagine that this could remove more cholesterol than lecithin from the corpuscles, the effect must be due to removal of some bodies in the plasma which have a protective action. Ponder<sup>27</sup> has shown that the proteins are the most important constituents in this respect.

As the amount of cholesterol in the blood is low in acholuric jaundice and is increased after splenectomy, this might be the cause of the change in fragility. That this is not the case is shown by the equally low cholesterol content of the blood in other types of anæmia where the fragility is not changed. It may be due to an increase of the lecithin, but of this at present we have no certain knowledge. Bolt and Heeres were only able to examine human blood from the splenic vein in a single case of hæmolytic anæmia,<sup>24</sup> when they found similar differences in the resistance of the red cells from the systemic and splenic veins. MacAdam and Shiskin<sup>26</sup> confirmed this difference in two cases of acholuric jaundice, but did not find it in a single case of splenic anæmia. It was found in the case here described, but Elliott was unable to confirm it.<sup>16</sup> These changes show why it is that normally splenectomy brings about an increased resistance of the red cells to hæmolysis and in acholuric jaundice

raises the abnormally low resistance nearer to the normal figure, for it seems probable that these two depend on the same factor. The increased resistance in the normal subject after splenectomy has been mainly established by the work of Pel<sup>7</sup> and Pearce,<sup>17</sup> and Larrabee has recorded an interesting case where the increased resistance lasted for thirteen years.<sup>28</sup>

#### *Discussion and Conclusions.*

The picture of acholuric jaundice is made up of increased fragility of the red corpuscles and of various signs and symptoms resulting from hæmolysis, which may be slightly, or from time to time considerably, in excess of what is normal. This excessive hæmolysis takes place mainly if not entirely in the spleen, removal of which, therefore, abolishes all symptoms of the disease. There is some evidence that the hæmo-lymph glands or accessory spleens may hypertrophy under these circumstances, but this does not reach such a degree as to reproduce the original picture.

That the symptoms of the disease are due to the hæmolytic activity of the spleen and not to the abnormal fragility of the red corpuscles is shown by the beneficial effects of splenectomy, for though it is true that the excessive fragility of the corpuscles is improved after operation, only very rarely is this improvement sufficient to bring about complete recovery of the red cells. Probably, therefore, when a familial case of acholuric jaundice develops during childhood, or even later as it often does, it is the activity of the spleen which has changed, and not the fragility of the red cells. We do not know of any case where this has been determined before the symptoms of acholuric jaundice have developed, and until this has been done the suggestion just made must remain a hypothesis. The opportunity to confirm this will arise when children are examined as a routine in families where some members have acholuric jaundice—a proceeding which we are now attempting to carry out.

In several cases the onset of acholuric jaundice seems to date from some acute illness, and the abnormal fragility of the red cells may well be the inherited weakness, which when acted upon by such extrinsic factors starts the spleen on its career of excessive hæmolysis. The improvement in the fragility of the red cells after splenectomy is similar to what is found normally, and certain factors in producing this change have been discussed. The improvement in the anæmia lessens the call for the production of immature red cells, which may show the abnormal fragility to a special degree; if this is so, it would be an additional reason for the improvement after splenectomy.

There are many interesting parallels between acholuric jaundice and pernicious anæmia—in fact the latter is sometimes difficult to distinguish from the “acquired” type of the former without an examination of the fragility. Here again splenectomy often brings about great improvement, but generally this is not maintained, which suggests that in acholuric jaundice the fragility of the red cells and the hæmolytic activity of the spleen together produce the symptoms, the fragility of the cells alone being relatively harmless, but that in pernicious anæmia the symptoms are due to the hæmolytic activity of the spleen and some unknown factor, which is itself sufficient to produce a fatal issue.

Splenic anæmia shows a closer clinical parallel to acholuric jaundice in the effects of splenectomy but not pathologically, for there is no excessive hæmolysis. Too little is known of the function of the spleen to be certain how the effect is produced, but it seems likely, as Osman concluded from a study of a large number of cases from this hospital, that “the syndrome is always the result of a chronic infection situated in the spleen itself.”<sup>25</sup> Our knowledge of patients with a large spleen from a long-standing malarial infection supports this view.

Removal of the spleen abolishes the symptoms of acholuric jaundice, without increasing the resistance of the red corpuscles to hæmolysis, much more than splenectomy normally does, and only very rarely, if at all, bringing the fragility back to normal. The abnormal fragility is probably the underlying inherited factor in the familial cases, the over activity of the spleen being started by extrinsic causes which lead to the full development of the disease.

We wish to thank Dr. Fawcett, under whose care these subjects were in-patients at Guy's Hospital, for his help and interest.

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# MASSAGE AND REMEDIAL EXERCISES IN MEDICINE

## PART III. MASSAGE AND REMEDIAL EXERCISES IN DISEASES OF THE LUNG AND PLEURA

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TREATMENT of diseases of the lung and pleura by physiotherapy may be considered under two headings: firstly, local treatment; secondly, general treatment. By the former is meant treatment applied to the chest itself; by the latter, treatment directed to improving the condition of the patient generally.

Local treatment has two main objects: to empty the bronchi or cavities in the lung; to promote the expansion of the collapsed or compressed lung.

### I. LOCAL TREATMENT

#### *Emptying of Bronchi or Cavities in the Lung*

One of the first essentials in treating a bronchus or cavity filled with mucus or muco-purulent secretion is to empty it as completely as possible. The secretion forms a nidus for the multiplication of bacteria, and until this is removed, the natural forces of the body do not have an adequate chance to exert their anti-bacterial activities. Treatment with this object is particularly useful in cases of chronic bronchitis, bronchiectasis or phthisis with cavity formation. It has been known for a long time that rubbing the chest with some form of liniment is of value in such cases; witness the traditional treatment of bronchitis by rubbing with camphorated oil. It is doubtful whether the oil itself has any effect, but there is no question that massage of the chest in the various forms described below excites coughing, and so promotes the evacuation of secretion; it is, of course, more effective if the patient lies in such a position that the force of gravity aids the passage of mucus to the trachea.

#### *Promoting the Expansion of Collapsed or Compressed Lung*

The principles of treatment on these lines consist firstly in making the patient breathe as deeply as possible, with a view of getting air into the collapsed alveoli; and secondly, in placing

him in such an attitude that the expansion of the healthy lung is limited, and that of the diseased lung free.

This is applicable to a large number of conditions; among them are cases where the lung has been compressed by a pleural effusion, clear or purulent, and cases where small portions of the lung are collapsed as the result of obstruction to the smaller bronchi, such as occurs in bronchitis.

In discussing local treatment the following conditions must be considered: (1) pleurisy; (2) fibrosis of the lung; (3) unresolved pneumonia; (4) bronchitis and bronchiectasis.

### (1) *Diseases of the Pleura*

There is no scope for massage or exercises in the acute stage of dry pleurisy; indeed any treatment directed to increasing the movements of the diseased side will aggravate the pain and probably make the inflammatory process more active. In cases of pleurisy with effusion, however, much can be done by physiotherapy. Whether the effusion is clear or purulent, it is apt by compressing the lung for a long period to cause fibrosis. The object of the treatment is to expand the diseased lung and so prevent fibrosis taking place. The more vigorous the treatment the better will be the lung expansion obtained, but there are other considerations which limit the amount of exercise desirable. These differ according to whether the effusion is clear or purulent, and it is convenient to consider the two conditions separately.

(a) *Treatment where the effusion is clear.*—Evidence continues to accumulate that in the vast majority of cases of clear effusion which are not secondary to cardiac or renal disease, the invading organism is the tubercle bacillus. In such cases the primary consideration in treatment is to avoid anything that makes the inflammation more active, and it is much better to run the risk of imperfect lung expansion than that of stirring up an infection which is quiescent. Any signs of active infection contraindicate any remedial exercises, and treatment must therefore be postponed until the temperature and pulse-rate have reached normal. Even after this has happened, treatment must be begun very carefully and advance extremely slowly. I have seen considerable harm result where this precaution has not been taken, and I remember one case of pleural effusion without any symptoms of lung involvement, where three days' injudicious treatment was sufficient to start a pyrexia which only subsided after two months.

The scheme of exercises given below is equally suitable to cases of clear or purulent effusion, but in the former progress as a rule must be much more gradual than in the latter.

The following rules, although not absolute, will be found useful in deciding on the severity of the exercises to be used in tuberculous cases.

(1) The exercises must be stopped if they are followed by any rise of temperature above the normal, or by any increase in the difference between the morning and evening temperature.

(2) If the pulse-rate taken from day to day shows a tendency to rise, exercise must be stopped, or at any rate reduced.

(3) Any increase in the amount of effusion, or the development of any signs or symptoms indicating advance in the involvement of the lung, is an absolute bar to any physiotherapy.

If treatment is carried out with these precautions, there is very little risk of it doing any harm, and I am quite satisfied, in the cases I have seen, that it does hasten the absorption of an effusion, and improve the expansion of the lung.

(b) *Treatment in cases of empyema.*—Of course no treatment can be done in cases of empyema until after operation for drainage of the pleural cavity, but once this is established, the exercises should be started as soon as possible. I have been immensely impressed by the difference in the results obtained in cases treated with and without physiotherapy, particularly if this is begun early. Patients are sometimes sent for exercise treatment months after operation, when fibrosis has already taken place in the underlying lung. Although improvement can be obtained in many cases, the results are not nearly so good as when treatment is started earlier. Others come with marked deformity of the chest, and an associated scoliosis; such deformities can be reduced, but their presence indicates so advanced a degree of fibrosis that little or no alteration in the lung can be hoped for. Scoliosis ought never to be allowed to occur as a complication of empyema, for it can always be prevented by exercises if these are not started too late.

Patients with uncomplicated empyema, who are not suffering severely from toxæmia, can start breathing exercises forty-eight hours after the operation, and in many cases I have ordered them within twenty-four hours. The severity of the scheme should be increased rapidly, and at the end of the first week after operation, if the patient's general condition is good, the hardest exercise should be sufficient to make the patient slightly short of breath. As time goes on, a greater degree of breathlessness should be aimed at, for the greater the dyspnoea the greater the expansion of the lung. Provided drainage is adequate, I have never seen any signs of toxæmia follow treatment on these lines.

In some cases, however, drainage is not perfectly satisfactory,

and pus tends to loculate. Under these conditions the temperature begins to go up, and the question arises as to whether exercise should be stopped. Personally, I think it should be continued, unless the patient shows serious signs of septic absorption, for the effect of exercise is to increase the amount of pus, and what usually happens is that the adhesions confining it break down, and pus is discharged, with consequent fall of temperature. Even if the adhesions do not break down, the increase in the amount of pus makes it easier for the surgeon to locate it.

Since the object of all exercises is to increase lung expansion, it is obvious that the best results cannot be obtained if the movements of the chest are restricted by tightly applied dressings. During treatment the chest should be uncovered and free of bandages, the wound being protected by a sterile pad secured by strapping or one or two turns of bandage. If possible the drainage tube should always be removed during treatment; its presence causes pain, and it is probable that by its mechanical irritation of the pleura it reflexly limits the movements of the effective side, and so hampers lung expansion.

A study of the movements and positions in the scheme of treatment given below shows that an attempt is made to limit the movements of the ribs in order to increase the expansion on the diseased side. It has been argued that limiting the movements of the ribs does not necessarily diminish the volume of air entering the lung on full inspiration, for it is possible that the greater descent of the diaphragm may compensate for the diminished movements of the ribs. A few x-ray observations have been carried out on normal subjects to determine this according to the following method.

The subject put the right hand on the head, bent the body over to the left and pressed the left arm firmly against the side of the left chest. A photograph was then taken at the end of a full inspiration (position A). The position was then reversed; the left hand being placed on the head, and the right arm being pressed against the right chest (position B). It was found that the volume of the right lung was greater in position A than in position B. It would have been satisfactory to have made similar observations in a case of empyema, but the difficulty of marking out the margins of the lungs exactly in such a case renders any measurements of size very liable to be inaccurate, and no definite conclusions can be drawn from them. If, however, by placing the patient in a suitable position, we can diminish the expansion of a lung in health, it seems reasonable to suppose that the same can be done if the opposite side is diseased.

### (2) *Fibrosis of the Lung*

Here the object of treatment is to expand the diseased portion of the lung as far as the fibrosis will allow. Of course this is impossible if the lung tissue has been completely replaced by fibrous tissue, but where this replacement is only partial, some improvement in expansion will often result. In selecting cases for treatment it is of the utmost importance to distinguish the tubercular from the non-tubercular cases. In the former, excessive exercise may easily light up quiescent infection, and it is therefore unwise to give even breathing exercises if the patient is febrile, or if the pulse-rate taken in the morning is rapid. It is impossible to lay down an absolute rule as to what should be considered a rapid pulse-rate, but it is safe to say that a rate of 90 or over is a contra-indication to physiotherapy of any kind.

Space does not permit of full discussion of considerations which should guide us in prescribing the amount of exercise for a tubercular patient; the effect on the patient's general condition affords the most important indication for treatment, and any tendency to a rise of temperature or persistent increase of pulse-rate shows that the exercise has been excessive, and indicates the need for curtailing it or stopping it altogether.

In non-tubercular cases there is much less danger of lighting up an infection, and, as in the case of empyema, vigorous work can be prescribed, provided it does not cause exhaustion.

### (3) *Unresolved Pneumonia*

Everyone is familiar with cases of pneumonia where physical signs of consolidation, complete or partial, persist for weeks after the acute stage of the illness is over. Here again massage over the affected lobe is used, though it is difficult to say how far it loosens the secretion in the alveoli. Of greater value is treatment directed to expanding the diseased lung.

### (4) *Diseases of the Bronchi*

It is in these diseases that reflex stimulation of the chest by the various types of chest massage is most useful. If the patient is sufficiently well to be out of bed, he should lie on his chest with pillows so arranged that the head is at a lower level than the body; the back of the chest is then vigorously massaged. In a case of bronchiectasis recently treated at Guy's, enormous quantities of secretion were expelled by this means.

Patients not sufficiently well to stand this position should

lie first on one side for treatment, and then on the other, in order to drain each lung separately.

In addition, exercises similar to those used in cases of empyema are given to expand the diseased lung, and these are of value in cases where the fibrosis is not too extensive.

## II. GENERAL TREATMENT

In all disease of the lungs or pleura, particularly in the case of the former, the patient suffers from some degree of breathlessness on exertion, and graduated exercises play an important part in improving the exercise tolerance. Enough has been said about the progression of the exercises in tubercular cases; in cases where the patient is suffering from fibrosis of the lung, or chronic bronchitis and emphysema, the treatment depends very largely on whether the circulatory system is healthy. In cases where there is myocardial degeneration, the treatment will follow the lines laid down in a previous article of this series.\* Where the heart is free from disease and the blood vessels healthy, exercises should be given which produce a slight amount of dyspnoea. These are gradually increased in severity, and it is found that as improvement takes place, the patient is able to stand harder exercises with less discomfort. A scheme similar to that already described in cases with a functional disorder of the heart can be used, but as a rule it is desirable to aim at obtaining rather less rise of pulse-rate than is advocated in the latter type of case; the hardest exercise should raise the pulse-rate to about 130 in young people, and to about 100—110 in older subjects.

### METHODS OF GIVING MASSAGE AND EXERCISES

It is convenient in describing the actual methods of massage and exercises first to outline the different varieties used in local and general treatment, and then to give in detail a scheme combining the two, which can be adapted with modifications for the various lung diseases mentioned above.

#### *Local Treatment*

The various types of local treatment of the chest can be described under the following headings :

- (A) Methods of massage.
- (B) Types of breathing exercises.
- (C) The position of the patient.

#### (A) *Methods of massage used in lung disease.*

- (1) "Chest clapping." This is a series of sharp blows struck with the flat of the hand, the wrist being loose.

\* G. H. Hunt : *Guy's Hosp. Rep.*, lxxv, 1925.

(2) "Chest hacking." The chest is struck a series of blows with the ulnar surface of the hands.

(3) "Chest vibration." The hands are placed on the front and back of the chest, and a series of small quick shakes are performed. The mechanical effect of these movements is to produce cough, so loosening mucus and freeing the air passages.



FIG. 1.

Showing patient in the half lying position with the left arm placed so as to fix the healthy side of the chest.

(B) *Types of breathing exercises.*

(1) The patient is taught to inspire deeply, and to feel the part of the chest to be moved, by placing the free hand on it.

(2) The patient takes a deep breath, to the counting of the masseuse, and tries to make the inspiration longer and longer.

(3) The patient takes a deep breath and contracts the abdominal muscles while holding the breath.

(4) The patient takes a deep breath, and while holding it, raises the arms from the side to above the head and brings them back to the side.





FIG. 2.

Showing patient in the lying position, with the legs and body bent to the left, so as to expand the right lung. The operator is pointing to the part of the chest where she wishes the patient to try and get the maximum expansion (localised breathing). This is known as the "Whiting position."

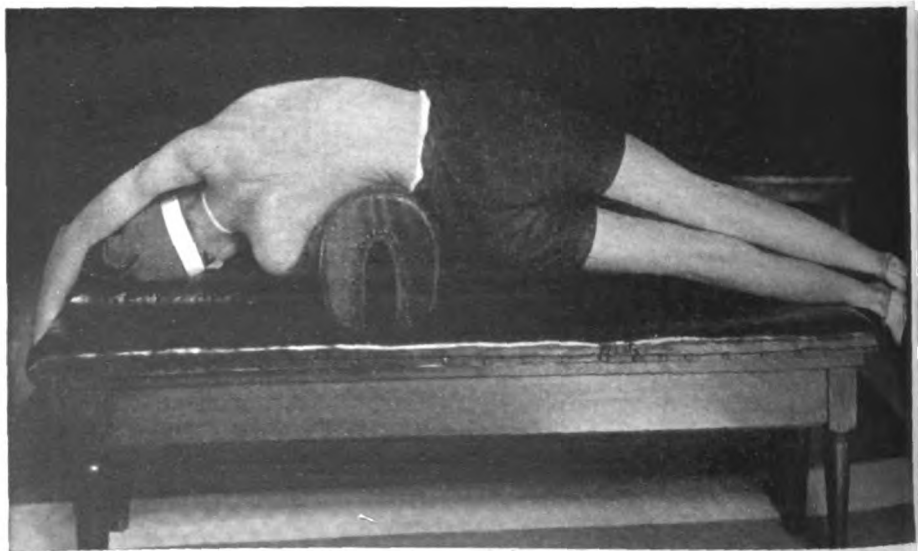


FIG. 3.

Showing the patient lying with the healthy side over a padded boom.

(C) *The position of the patient.*

Exercises are given with the patient lying or standing.

(1) *Exercises in the lying position for a patient with disease of the right lung.*

(a) *The patient lies on his back.*

The upper part of the patient's arm is pressed against the left side of the chest, and the operator places one hand over the patient's upper arm and the other over the wrist, which is held over the abdomen at the level of the umbilicus. The patient is then instructed in breathing with a view to getting expansion of the diseased lung, first trying to get the maximum movement at the side of the chest, then at the apex of the lung.

At first these movements are done with the patient's right arm to the side; later the arm is gradually raised until it is fully stretched above the head (Fig. 1).

This exercise is progressed by bending the trunk in the dorsal region towards the left side, and by carrying the legs towards the same side, the pelvis being kept flat (Fig. 2).

During the treatment, chest vibration, light hacking and shaking may be added to make the patient cough and so loosen mucus.

(b) *The patient lies on the healthy side with a firm pillow placed between the arm and the chest.*

The breathing exercises are repeated as with the patient lying on the back; the position of the free arm being altered in the same way.

(c) *The patient lies with the left side of the chest over a padded boom, the end of a couch or the arm of a chair, thus fixing the healthy side more firmly (Fig. 3).*

Breathing exercises are then repeated as above.

(2) *Exercises with the patient standing.*

The patient stands with the healthy side against some form of support. The left arm is kept steady to the side by holding on to a firm support; the arm on the diseased side is stretched well above the head and fixed. The breathing exercises are repeated as in the lying position (Fig. 4).

### *General Treatment*

The exercises are all double-sided and aim at improving circulation and respiration generally. As an introduction to exercises and in cases where the patient is too weak for any exertion, massage is given according to the following scheme.

The patient lies on a couch with pillows arranged to support his shoulders and head, and to flex the hip and knee; the

shoulders are allowed to fall backwards, and the chest is pushed forward.

Effleurage and kneading are given evenly and rhythmically

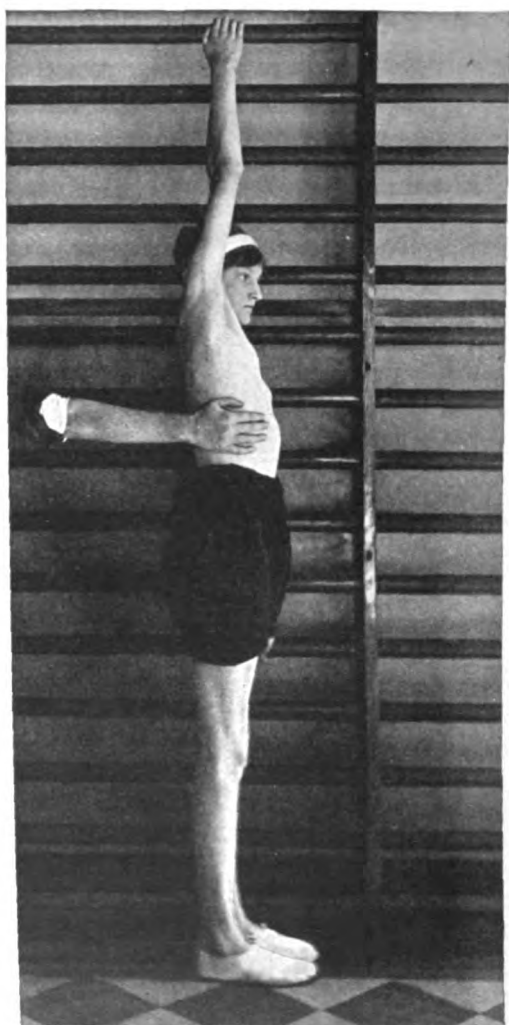


FIG. 4.

Showing method of fixing the healthy chest with the patient standing.

to limbs. The first treatment lasts ten minutes, and the time is increased up to thirty minutes. The massage is continued for a few days or several weeks, according to the condition of the muscles and of the lung.

*Exercises*

The following scheme of exercises is given to be done by the patient :

(1) *Exercises with the patient in the lying position.*

(A) Movements to the arm and leg.

These consist in flexion and extension of the joints, beginning with the smaller joints and progressing to the larger joints.

(B) Movements of the trunk.

The patient raises and lowers the body, inspiring deeply while raising the body, and expiring while lowering it.

(2) *Exercises with the patient in the sitting position.*

The limb and trunk exercises are repeated, and there are added the following exercises for the trunk :

(A) The trunk is rotated from side to side.

(B) The trunk is bent from side to side.

(C) The trunk is rotated and fixed. The trunk is then bent from side to side.

(D) The trunk is bent forwards and backwards, the movements involving all the joints of the vertebral column.

(3) *Exercises with the patient standing.*

All the previous exercises are repeated, and, later, walking, running, skipping, climbing, dancing and jumping are added.

The work done by the patient is made harder :

(a) By increasing the frequency of the movements.

(b) By increasing the rate of the movements.

(c) By increasing the duration of the exercises.

(d) By adding resistance.

The following progressive schemes of exercises are suitable in the treatment of lung conditions.

*Scheme 1*

(a) Half lying (1), apical breathing (Fig. 5).

(b) Half lying, double arm kneading (2).

(c) Half lying, breathing and chest vibrations.

(d) Half lying, double leg kneading (3).

(e) Half lying, deep breathing.

Scheme 1 is suitable for patients who are still very weak, e.g., for a patient twenty-four hours after an operation for empyema. As improvement occurs, exercises such as flexion and extension of the wrist and ankle, and later of the elbow and knee, are added. The movements at first are done six times in

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each direction, and the number is increased by one or two daily; at the same time larger groups of muscles are brought into action.

### *Scheme 2*

(Started at the end of one, two or three weeks.)

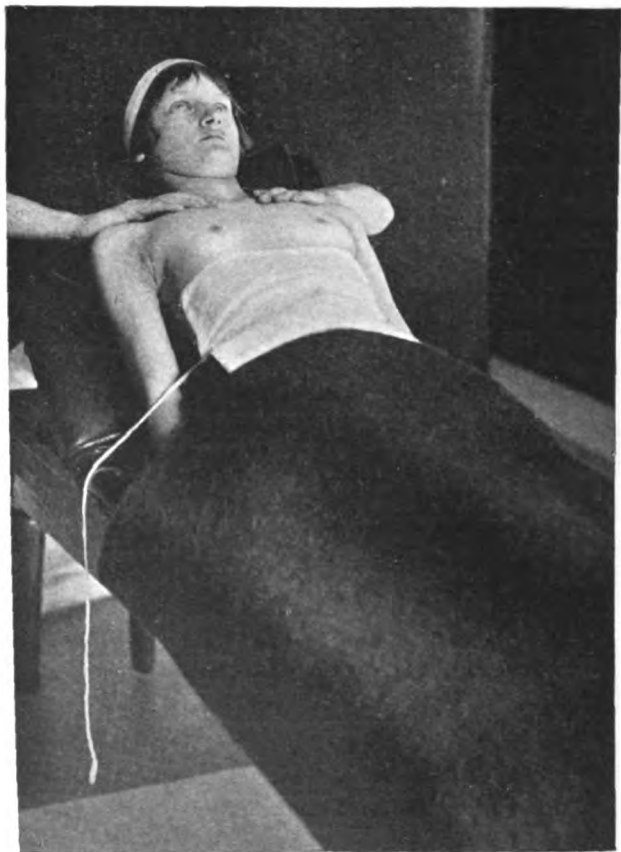


FIG. 5.

Showing the patient in the lying position.  
The operator is teaching apical breathing.

(a) Half stretch, half low grasp, side bend lying (4); deep breathing (fixing normal chest) (Fig. 2).

(b) Lying, alternate leg flexion and extension (bicycling movement). (Six times, and progress quickly.)

(c) Half neck rest, half low grasp, sitting (5); localised breathing (6) and chest clapping. (Clapping not given in empyema.)

(d) Sitting, double arm flexing and extending (7).

(e) Half neck rest, half low grasp, sitting (5); side bending to normal side (8) and breathing.

If there is no external wound, chest clapping and chest vibration may be used during the breathing exercises.

The number of times the exercise is performed depends on the effect, but a good working rule is for the patient to begin by doing each new exercise six times, and to increase the number of times by two a day.

### *Scheme 3*

(Started at the end of two, three or four weeks. The patient is still in bed.)

(a) Half stretch, side lying, deep breathing (Fig. 3).

(b) Sitting, double arm flexion and extension (7).

(c) Wing sitting (9); alternate trunk turning (10), with deep breathing.

(d) Arm lean sitting (11); chest clapping and hacking (if no external wound).

(e) Wing stoop sitting (12); trunk raising (vertebra by vertebra, starting with the lower part of the spine).

(f) Lying, alternate leg flexing and extending (quickly for two or three minutes).

(g) Half stretch, half low grasp, standing (13); side chest clapping (Fig. 4). (Omit chest clapping if external wound.)

This scheme may be progressed in various ways :

(a) By increasing the rate at which the exercise is performed.

(b) By teaching the patient to hold the breath for a longer period.

(c) By continuing the exercise for a longer period.

(d) By giving a greater weight to be lifted.

### *Scheme 4*

(The patient is able to get out of bed.)

(a) Half stretch, half low grasp, sitting (14); trunk side bending (8) and breathing. (Bend to normal side and progress breathing.)

(b) Walking (progress to running and skipping).

(c) Stretch, grasp, stride, standing, trunk flexing and extending (15) (quick pace).

(d) Half stretch, half low grasp, standing (13); side chest clapping (Fig. 4). (Patient standing with normal side against the wall.)

(e) Tailor sitting (16); quick alternate trunk rotation

(Let the arms follow the trunk to allow a larger range of movement with each rotation.)

(f) Sitting, double arm flexing and extending (7). (Lifting 2 lbs. or 3 lbs. and working for two, three, or four minutes.)

(g) Running up and downstairs (as scheme progresses).

(h) Standing, double arm raising sideways (17) and chest clapping.

From this time onwards, general free gymnastics may be employed, if possible in a class with other people.

Schemes for the treatment of bronchitis may be adapted from the above, with the addition of abdominal massage and breathing exercises for both sides of the chest.

*Explanation of the Terminology used in the Schemes*

1. *Half lying.* Lying with three or four pillows.
2. *Double arm kneading.* Kneading of both arms.
3. *Double leg kneading.* Kneading of both legs.
4. *Half stretch, half low grasp, side bend, lying.* The patient lies on the back, with a firm pillow placed between the arm and chest.
5. *Half neck rest, half low grasp, sitting.* The patient sits on a stool, the hand of the healthy side placed on the back of the neck, and the other grasping the stool, the arm being kept close to the side.
6. *Localised breathing.* Breathing exercises for the expansion of some particular part of the chest (apex or side).
7. *Double arm flexion and extension.* The arms are flexed with the hands placed on the shoulders; the arms are then raised above the head, and brought down to the side.
8. *Side bend to normal side.* The trunk is bent towards and away from the normal side.
9. *Wing sitting.* Sitting with both hands resting on the hips.
10. *Alternate trunk turning.* Rotating the trunk first to one side and then to the other.
11. *Arm lean, sitting.* The patient sits, and the trunk is bent forward with the backs of the hands resting on the forehead, and the head and hands resting on some form of support (e.g., a couch).
12. *Wing stoop, sitting.* The patient sits with the hands on the hips, the trunk bent forward.
13. *Half stretch, half low grasp, standing; side, chest clapping.* The patient stands with the hand of the healthy side grasping some low support; the other hand is stretched above the head (Fig. 4).

14. *Half stretch, half low grasp, sitting.* The patient sits with the healthy side fixed by grasping a low stool, the other arm is stretched above the head.

15. *Stretch, grasp, stride, standing; trunk flexion and extension.* The patient stands with the feet apart, the arms stretched above the head and the hands clasped, and flexes and extends the spine.

16. *Tailor sitting.* Sitting on the floor with the legs crossed in front (as a tailor).

17. *Double arm raising sideways.* The arms are held to the side fully extended, and raised to the horizontal and brought to the side again.

In conclusion, my best thanks are due to Miss Angove, Sister-in-charge Massage Dept., Guy's Hospital, for preparing the scheme of massage and exercises. I must also thank Miss Looker for taking the photographs used in this article.



## DIVERTICULA OF THE COLON

By A. F. HURST, M.D., Physician to Guy's Hospital, and  
R. P. ROWLANDS, M.S., Surgeon to Guy's Hospital.

THE earliest description of diverticula of the colon is that published by Cruveilhier <sup>1</sup> in 1849. In the English and American literature on the subject his important work appears to have been entirely overlooked. Curiously enough, Virchow <sup>2</sup> is generally credited with the first account of the condition, although on looking up the reference we find that the *diverticula coli* about which he wrote are simply the haustra of the colon which bulge out between the longitudinal bands, and he makes no reference whatever to the pathological condition now known as diverticula. According to some French authors Morgagni described diverticula of the colon as long ago as 1796; here again we find that the authors must have consulted his work without any knowledge of the language in which he wrote, as the "diverticulum" of the colon to which he refers is the segment of the bowel projecting into a Littré's hernia.

In his *Traité d'Anatomie Pathologique* Cruveilhier gives the following admirable analysis of the anatomical characters of diverticula of the colon :

"It is particularly in the rectum and sigmoid that hernias of the mucous membrane through the muscular coat ('hernies tuniquaires') are observed. The cause of this predilection lies in the unequal resistance offered by the walls of the colon, the longitudinal fibres being collected in three parallel bands, which offer a considerable resistance in comparison with the relatively weak parts between them. Moreover, the rectum and sigmoid act as a store-house for fæcal material, upon which the main force of the act of defæcation is expended. For these two reasons it is natural that the sigmoid should be specially liable to develop this form of hernia.

"It is among old men, and especially those who were subject to constipation, that one not infrequently finds between the bands of longitudinal muscle fibres in the sigmoid a series of small, dark, pear-shaped tumours, which are formed by hernias of the mucous membrane through the gaps in the muscular coat. Their dark colour is caused by small and very hard accumulations of fæces within them. These little sacs, the shape of which is sometimes oblong like the finger of a glove,

are often hidden in the fat which covers the large intestine and thus escape a cursory examination. The largest number I have ever seen were in the body of Professor Alibert : there were several hundred, each with a narrow and distinct orifice. One can imagine that these little mucous sacs may become irritated by the fæcal material they contain and thus become the seat of inflammation and perforation."

Though Bristowe <sup>4</sup> had shown a typical specimen of multiple diverticula of the colon to the Pathological Society of London in 1855, the first author to describe the condition in the English language was Habershon, <sup>5</sup> physician to Guy's Hospital, who in 1857 wrote the following excellent account of diverticulosis :

"Pouches of the colon sometimes become of a considerable size; generally the circular fibres of the canal surround them, but not very unfrequently the circular fibres yield, and the mucous layer projects, covered only by the peritoneum, forming a more elongated sac, filled with mucus, or more frequently fæces. The orifices of these small sacs are bounded by the hypertrophied and longitudinal fibres, and their contents remain almost shut off from the intestinal canal. These pouches are the result of constipation, the muscular fibres become hypertrophied, but their effort to propel onward their contents leads to these minute hernial protrusions.

"I have most frequently observed them in connection with the sigmoid flexure; but they probably occur at any part where the longitudinal fibres form a triple band rather than a uniform layer. In one case they were situated about every half-inch, forming a double row on each side of the colon. No muscular fibres could be detected in several of them, beyond the immediate vicinity of the mouth of the sac, but merely mucous membrane, submucous cellular tissue, fat and peritoneum. These pouches do not appear to produce any symptoms, or lead to dangerous result.

"*Case CXXVII.*—A remarkable case of this kind I observed in a patient, aged 62, who died from cancerous disease of the liver and lungs, bronchitis and emphysema. The sigmoid flexure and rectum were contracted, and presented numerous pouches, some of which were half an inch in length; they were arranged in two rows about one inch apart: these pouches consisted of mucous membrane and peritoneum; the circular muscular fibres were placed between the pouches, and the longitudinal fibres on either side, and both were hypertrophied. The pouches were filled with mucus and fæces. There was no ulceration or evidence of cicatrix, but it appeared that the constipated bowels to which the patient had been subject had

led to unequal pressure and saccular distension, or herniæ of the mucous membrane."

In 1885 Lane <sup>6</sup> described in these *Reports* a specimen of diverticula in a partially obstructed loop of colon found in a hernial sac and associated with diverticula of the bladder; the paper is of special interest, as it contains the first published illustration of the condition (Fig. 1).

These early observations attracted very little attention, and it was not until the work of Beer <sup>7</sup> in 1904 and of Maxwell Telling, <sup>8</sup> who in 1908 collected 83 cases from the literature and added 24 new ones, that the pathological and clinical importance of the condition at last became generally recognised. In 1917

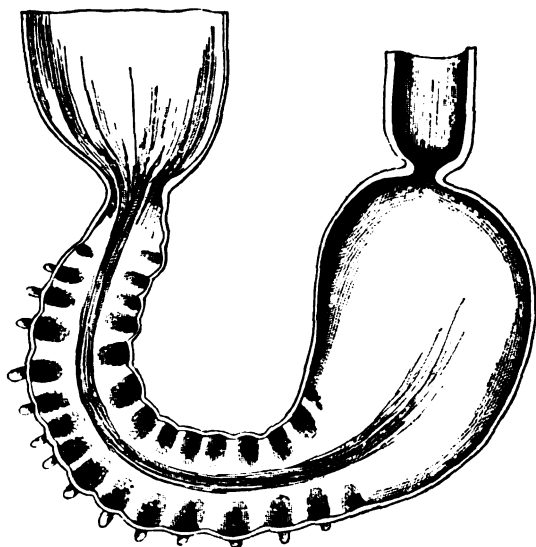


FIG. 1.

Diverticula of a loop of colon found in a hernial sac. Reproduced from the illustration of a paper by Sir W. Arbuthnot Lane in the *Guy's Hospital Reports* for 1885.

Maxwell Telling published with Gruner a further paper founded on the study of 324 recorded cases.

Up to 1913 diverticula of the colon were always an accidental discovery at operation or post-mortem. In that year de Quervain <sup>9</sup> in Switzerland and subsequently one of us (A. F. H.) in England and Case in America diagnosed the condition with the aid of the x-rays. Radiology has since shown the frequency of diverticulosis, in which diverticula are present without producing symptoms, and has proved that their inflammation—diverticulitis—far from being a pathological curiosity as it was regarded for over fifty years after Cruveilhier's classical description, is really the most common cause of pain in the left iliac fossa.

The surgical aspects of diverticula of the colon have been predominant in the literature since 1907, when Mayo, Wilson and Giffin<sup>10</sup> reported five cases treated by operation, but the comparative ease with which the condition can now be diagnosed in its early stages has led to the gradual development of medical treatment, which should eventually render operation only necessary in exceptional cases.

#### ETIOLOGY AND PATHOGENESIS

In nearly all cases in which a clinical history is obtainable it is found that individuals with diverticula of the colon have suffered for many years from more or less severe constipation. The distribution of the diverticula corresponds with the position in which accumulations of hard faeces most commonly occur. Thus, apart from diverticula of the appendix, they are rarely found above the middle of the descending colon, though one out of Mayo's first series of forty-two cases occurred at the hepatic flexure and one in the proximal part of the transverse colon, and they increase in number and size as the middle of the pelvic colon is approached. Although in dyschezia faecal accumulation occurs first in the rectum, this is very rarely the seat of diverticula, owing probably to the uniform thickness of its muscular coat, but Mayo has operated on two rectal cases and on one in which the diverticula originated in the anal canal.

The average age of eighty cases of diverticula collected by Telling was sixty, the youngest being a constipated woman of twenty-two, but Hartwell and Cecil have more recently published a case occurring at the age of six; the average age of one hundred and eighteen cases in the Mayo Clinic between 1907 and 1924 was fifty-five (Judd and Pollock<sup>11</sup>). Of 118 cases 71 per cent. were men and 29 per cent. women.

Thus diverticula are very rare before the age of forty, although very severe constipation is not uncommon among quite young people, and they occur much more frequently in men than in women, although, until old age approaches, the latter are more subject to constipation; hence other factors must be concerned in their production in addition to constipation. Chief among these are muscular atrophy and the presence of excessive fat in the walls of the colon. Owing to the atrophy of the muscular layer of the colon which occurs in old age, pressure from within produces diverticula more readily than in earlier life. The rare cases of diverticula in younger individuals may perhaps be associated with that form of constitutional constipation which is probably due to deficient development of the intestinal musculature. Frequently well-marked obesity

is present, and in other cases the patients have been fat, but emaciation has occurred at a later period. In the obese there is an excessive development of the appendices epiploicæ and also of fat under the serous coat of the intestine. The presence of fat diminishes the resistance of the intestinal wall to pressure from within, so that diverticula are likely to occur, especially into the appendices epiploicæ. They are still more easily produced if an excessive development of fat has for any reason rapidly disappeared.



FIG. 2.

Single diverticulum of descending colon, without symptoms, shown after administration of opaque enema.

The blood vessels of the colon are surrounded by a small quantity of lax connective tissue where they pierce the intestinal wall. Consequently points of diminished resistance are produced, which, next to the appendices epiploicæ, form the most frequent site of diverticula.

#### MORBID ANATOMY

In the majority of cases a large number of diverticula are present, but occasionally only a single one is found (Figs. 2 and 3).

They are generally arranged in two rows, and are often confined to the appendices epiploicæ, but sometimes a few or all may be situated close to the mesenteric attachment. They are rarely widely disseminated, being generally confined to a segment of the colon not exceeding 10 to 20 cm. in length or at most 30 to 40 cm. Some are so small that they are barely visible to the naked eye, whilst others attain a diameter of half an inch.

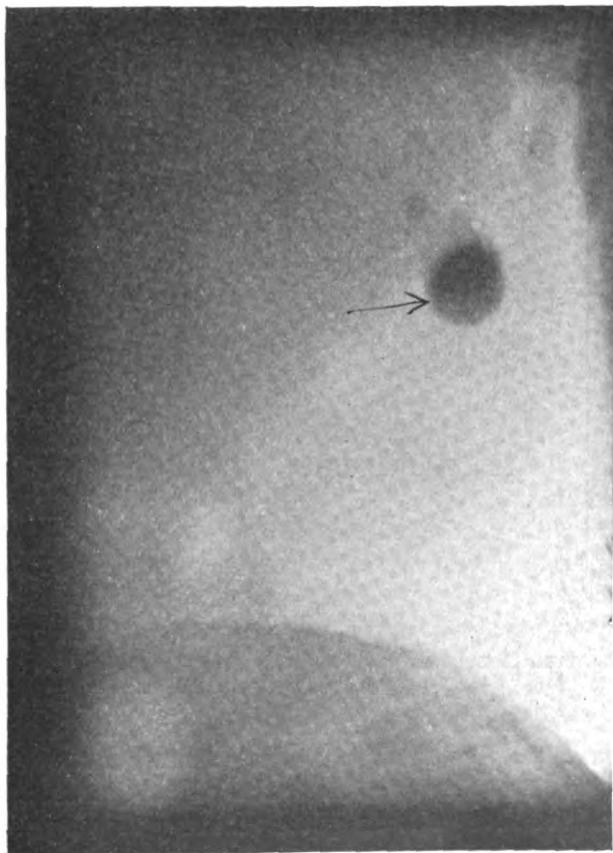


FIG. 3.

Same case as Fig. 2; single diverticulum still filled 24 hours after evacuation of opaque enema.

They very rarely become larger, as secondary pathological changes interrupt their growth. The smallest are semi-globular in shape. The larger ones consist of fusiform pouches; in these the lumen communicates by a very narrow neck with the lumen of the bowel.

The walls of the diverticula are often formed of mucous membrane alone, as they have "blown out" where the muscular

support was absent. In other cases all the coats of the colon may be represented in the walls of the smaller diverticula, but as they grow larger the muscular layer gradually disappears, and the mucous membrane frequently becomes atrophied. The sharp division formerly made between true diverticula, in which all the coats are represented, and false diverticula, formed of the mucous membrane and peritoneum alone, is thus incorrect.

The diverticula are almost always filled with hard *faeces*. Owing to the deficiency in their muscular coat and to the narrowing of the neck which joins them to the bowel, their contents can only be expelled with difficulty. There is a constant tendency for them to increase in size owing to further quantities of *faeces* being squeezed into them from the lumen of the gut whenever it is distended with *faeces*.

### RESULTS

Diverticula of the iliac and pelvic colon give rise to no symptoms unless pathological changes occur in them. Owing, however, to the stagnation of *faecal* material within them, they are very liable to inflammation. Ulceration takes place, and as a result of the thinness of their walls their peritoneal covering becomes infected. The wall of the bowel is thickened, the mesentery thickened and shortened, and adhesions form, especially to the bladder.

The irregular sausage-shaped tumour formed by the inflamed colon has been frequently mistaken for cancer, especially when symptoms of chronic or acute obstruction have been present. In very chronic cases the development of fibrous tissue produces a tumour, which even during an operation or at the autopsy has been mistaken for a growth, until microscopical examination showed its inflammatory origin and careful dissection revealed the presence of long and tortuous diverticula in the mass. In other cases the tumour has been left as inoperable, but the subsequent complete recovery of the patient has revealed its true nature.

### SYMPTOMS AND DIAGNOSIS

The mere presence of diverticula of the colon—the condition to which de Quervain<sup>9</sup> has given the name of diverticulosis—gives rise to no symptoms. It is often discovered in the routine x-ray examination with a barium enema of patients suffering from constipation and other intestinal disorders. If systematically looked for in constipated but otherwise healthy individuals over the age of forty, diverticulosis would probably be found to be a quite common condition. Its sole importance is that

under certain conditions the diverticula become inflamed and the condition of diverticulitis results.

The earliest descriptions of the clinical aspects of diverticulitis were published by surgeons, who had operated upon advanced cases which had given rise to serious symptoms. Since it has become possible to diagnose the condition with comparative ease by means of the x-rays, it has gradually become recognised that a mild form of diverticulitis is not uncommon, and that it gives rise to much less obvious and severe symptoms than those which were originally described. Many cases of intestinal dyspepsia, often diagnosed vaguely as colitis, are really due to diverticulitis. In any patient over the age of forty or forty-five, who complains of attacks of discomfort in the lower part of the abdomen, and especially in the left iliac fossa, associated with constipation, irritability of the bladder, and occasionally with slight pyrexia, the possibility of diverticulitis should be considered. In such cases there is always definite tenderness in the left iliac fossa and often on deep pressure just above the pubes. In the early stages the iliac colon may appear to be slightly thickened, but no definite tumour is present. But a tender tumour in the left iliac fossa is much more frequently due to diverticulitis than a growth, the iliac colon being an unusual situation for carcinoma, whereas it is affected either alone or with the pelvic colon and occasionally the descending colon in a large majority of cases of diverticulitis.

In more advanced cases the symptoms are those of an acute inflammatory condition simulating appendicitis, but on the opposite side, or of chronic intestinal obstruction simulating cancer of the colon.

(a) *Inflammatory type*.—Acute inflammatory attacks may occur, which are exactly similar to those of appendicitis, except that the local signs are on the left side. In cases of doubt the x-rays not only show the presence of diverticula, but they visualise the appendix and show that it is in its usual position; at the same time the tender area is found to be over the iliac and pelvic colon and not the appendix. The attacks may begin quite suddenly with acute pain after a heavy meal, whilst straining at stool, and after an aperient or during the administration of an enema, all of which produce active peristalsis of the colon, which might tear adhesions formed by inflamed diverticula. In other cases pain has occurred whilst at work, lifting a weight, jumping, hunting, or travelling in a jolting motor-car, and acute symptoms have developed after an operation on some other part of the abdomen.

All degrees of inflammation occur. Sometimes perforation



takes place with the formation of a localised abscess, which may discharge into the bowel, vagina or bladder, sometimes with the formation of a temporary or permanent fistula, but especially a vesico-colic fistula. In others the abscess, or the original perforation, opens into the peritoneum and sets up a spreading peritonitis. One of us (R. P. R.) has operated on three cases of late diffuse peritonitis due to this cause, with two deaths. It is important to remember diverticulitis as a possible cause of peritonitis of obscure origin.

(b) *Obstructive type*.—Diverticulitis often produces symptoms almost indistinguishable from those of cancer of the colon. In 1906 Moynihan<sup>13</sup> showed that fibrous stenosis of the colon due to diverticulitis may mimic cancer of the colon. One of us (R. P. R.) published two cases of chronic obstruction of the pelvic colon due to this cause.<sup>14</sup> The specimen successfully removed from one of the cases is in the Museum at Guy's Hospital; it shows very well the presence of diverticula along the mesenteric border with perforation into the mesentery, which is occupied by a mass of inflamed tissue with necrotic areas and small abscesses. A great many cases of local inflammation of the colon, with or without obstruction and due generally to diverticulitis, have in the past been considered to be due to malignant disease. It is therefore of vital importance to make microscopical examinations of supposed growths of the colon, and also to examine all specimens for diverticula. It is often not at all easy to find these, even when the bowel is laid open. Sometimes they can be found only by the careful use of a probe. It is probable that patients who survive more than four years after a colostomy for supposed growth of the pelvic colon are not really suffering from cancer, but from simple stenosis due to inflammation extending from diverticula.

The sigmoidoscope is useful in excluding a growth of the rectum or lower six inches of the pelvic colon. In some cases the lumen of the bowel is seen to become progressively more narrow and more or less fixed at a point proximal to the pelvi-rectal flexure. The mucous membrane is generally normal in appearance. Occasionally it is red and swollen in the affected region, but it never bleeds on simple contact with the instrument and it is never ulcerated, as it frequently is immediately below a malignant stricture, because the inflammation is generally confined to the outer coats of the bowel and to the mesentery. Diminution in the lumen of the bowel and swelling of the mucous membrane may make it difficult to see the openings into the diverticula; they are also likely to be hidden by folds of mucous membrane, but Bensaude<sup>15</sup> and

others have seen them in a few cases. In one case of Mayo's the tumour had become partly intussuscepted into the rectum and was seen with the sigmoidoscope.

The stools rarely contain any pus or blood recognisable by the naked eye or microscopically, and occult blood is generally absent. This is a very important point in diagnosis, as, even in the absence of obvious blood, occult blood is always present both in primary cancer of the colon and in cancer associated with diverticulitis.

In most cases pyrexia and a moderate degree of polymorpho-

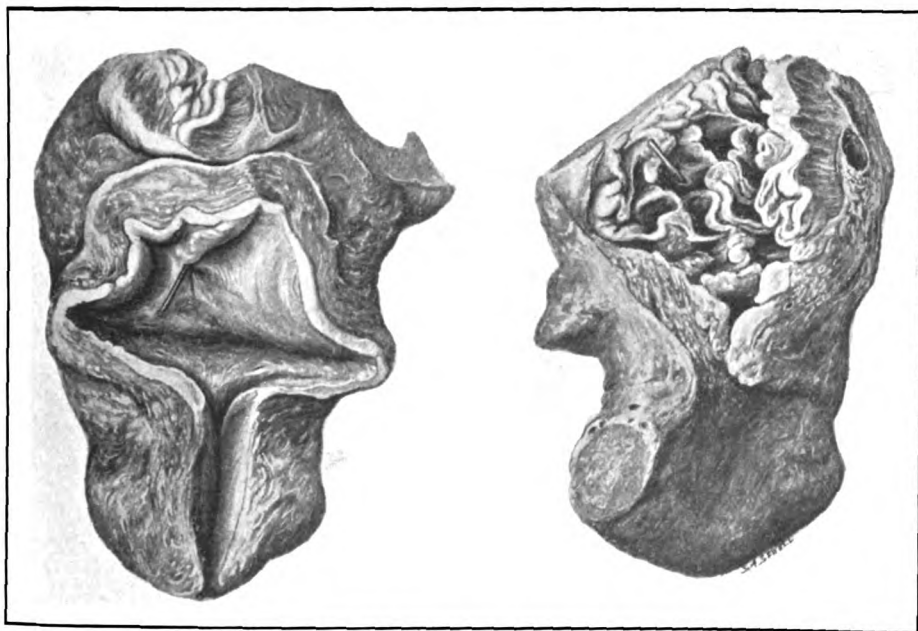


FIG. 4.

Specimen of vesico-colic fistula from Guy's Hospital Museum. A piece of colon with numerous diverticula, burrowing in 2 ins. of inflammatory tissue which separates it from the back of the bladder; one diverticulum, communicating with a small abscess cavity, has ruptured into the bladder. The patient, a man of 65, was under Thomas Bryant in 1882; he had passed wind for 12 years and faeces for 6 months *per urethram*. (Reproduced, by permission, from the *British Journal of Surgery*).

nuclear leucocytosis are present, both being comparatively rare in cancer. The secondary inflammation results in more pain than is common in growth apart from the colic due to obstruction. The long duration of symptoms with little or no wasting, cachexia or anæmia, and the frequent history of life-long constipation are further points in favour of diverticulitis.

In 19 out of 118 cases of diverticulitis on which operations were performed at the Mayo Clinic between 1907 and 1924, 19

also had carcinoma of the colon, but in several the association was obviously accidental, and Judd and Pollock believe that a patient with diverticulitis is no more likely to develop cancer than an average individual. In some cases a growth in the rectum or lower end of the pelvic colon has led to partial obstruction, and the increased internal pressure in the segment of bowel immediately proximal to it has doubtless been an important factor in the pathogenesis of the diverticula.

#### VESICO-COLIC FISTULA

Frequency of micturition and discomfort in the bladder are common symptoms of diverticulitis, and their association with other signs of intestinal disorder should suggest the likelihood of this diagnosis. The bladder irritability is doubtless due to adhesions between the inflamed colon and bladder. In the presence of such adhesions an ulcerated diverticulum or an abscess which has formed in connection with one may rupture into the bladder (Fig. 4). Cripps<sup>16</sup> was the first to show that a vesico-colic fistula results much more frequently from inflammatory changes in the colon than from cancer. A case published by Sydney Jones<sup>17</sup> in 1858 proved that ulceration and perforation of a diverticulum might produce a vesico-colic fistula, and the investigations of Telling proved that this is its usual cause. One of us (R. P. R.) has operated on several cases of the kind.

#### RADIOLOGICAL DIAGNOSIS

The x-rays afford the only means of recognising the presence of diverticula of the colon with certainty. Both an opaque meal and an opaque enema should be used; although the latter is much more likely to demonstrate the presence of the diverticula, the former helps to determine what effect they are exerting on the normal intestinal activity. In many cases the diverticula escape recognition after the opaque meal, but the degree and localisation of any delay in the passage through the colon, the exact situation of any area of tenderness, and the presence of fixation caused by adhesions can be studied. In Fig. 5 a very early stage of diverticulosis is shown; this was accidentally discovered during the radiological examination of a man of fifty-five with a jejunal ulcer. When an opaque enema is given, it may be held up temporarily or permanently in some part of the pelvic or iliac colon, showing that a considerable degree of obstruction is present, or, if it passes without difficulty, any local or widespread diminution in the lumen of the bowel is recognised. It is important to remember that a barium enema may give an exaggerated idea of the degree of obstruction,

as the narrowing caused by the contraction of newly formed fibrous tissue and by the presence of inflammatory material may be greatly increased by spasm; the latter is only temporary and is likely to be increased by the mechanical stimulus caused by the distension following the injection of the enema. If there



FIG. 5.

Iliac colon visualised 24 hours after an opaque meal.  $D^1$  shows the earliest stage in the development of a diverticulum—a slight outward bulge between two shallow indentations.  $D^2$  shows a fully formed diverticulum with a narrow neck. Careful inspection shows the presence of several other diverticula in various stages of development. (Dr. P. J. Briggs.)

is any evidence of narrowing, whether organic or functional, it is clear that diverticulitis and not merely diverticulosis is present.

During the administration of the opaque enema the diverticula may be recognised. But their exact relations and their number can only be determined with accuracy if the patient is re-examined after the greater part of the enema has been

evacuated (Fig. 6). The diverticula remain filled, and there is generally still a small quantity of the opaque fluid present in the lumen of the bowel which is no longer distended. Two rows of small rounded shadows with narrow necks connecting them with the intestinal lumen can generally be recognised.



FIG. 6.

Diverticula of iliac and pelvic colon seen immediately after evacuation of the greater part of an opaque enema.

They are often still visible twenty-four hours or even several days after the enema has been given (Fig. 7).

#### TREATMENT OF DIVERTICULOSIS AND PROPHYLAXIS OF DIVERTICULITIS

If in the course of an x-ray examination an individual is found to have diverticula of the colon, their significance should be explained to him, and he should be given instructions in order

to prevent pathological changes developing in them. In this manner it should be possible to provide a true prophylaxis of diverticulitis, especially if middle-aged and elderly people suffering from intractable constipation were habitually subjected to an x-ray examination, so that the presence of diverticula would more often be recognised before they had become inflamed.

The chief object of treatment is to prevent stagnation of hard fæces from occurring in the diverticula. Whatever other treatment may be required for the constipation, it is essential to give sufficient liquid paraffin to keep the stools permanently

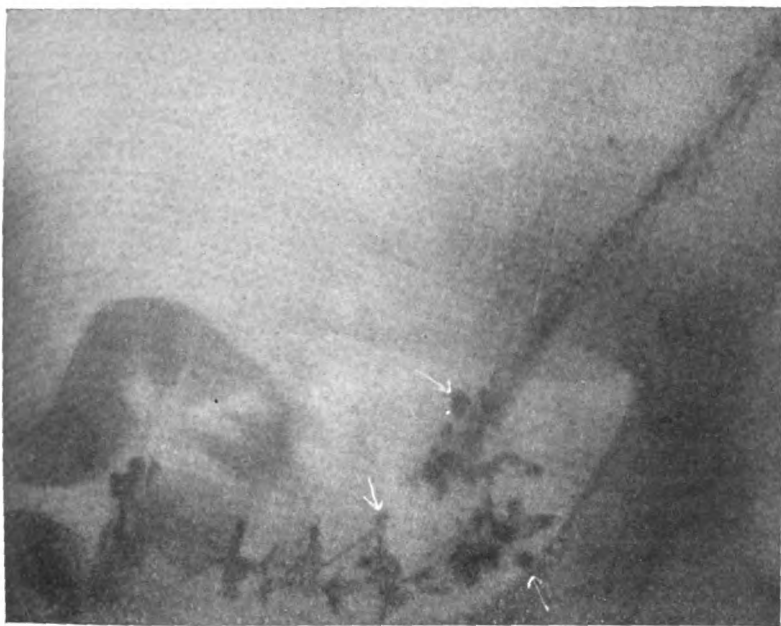


FIG. 7.

Diverticula still filled 24 hours after evacuation of opaque enema.

soft, without, however, causing actual diarrhoea. As a rule a tablespoonful taken morning and evening proves sufficient. In addition to this a diet should be chosen which leaves no solid undigested residue such as might become lodged in a diverticulum and cause local irritation. While, therefore, those articles of diet which stimulate peristalsis chemically should be taken in abundance, all salads, pickles and green vegetables, except in the form of fine purées, and all pips and skins of fruit, whether raw, stewed or in jam, and currants, raisins and lemon-peel in cakes and puddings must be absolutely prohibited. With treatment of this kind it is even possible that the diverticula may diminish in size and that small ones may disappear.

## TREATMENT OF DIVERTICULITIS

## (a) MEDICAL TREATMENT

The medical treatment of diverticulitis consists in complete rest in bed till active inflammation, as shown by the presence of pain, tenderness, muscular rigidity, pyrexia and leucocytosis, has disappeared. During this period—and also for the rest of the patient's life—the diet described under the head of prophylaxis should be given.

From six to eight ounces of liquid paraffin are run into the bowel by rectum every evening and are retained during the night. By this means hard fæces in the iliac and pelvic colon and in the diverticula are softened and evacuated the following morning, either spontaneously or after the administration of a pint and a half of warm water introduced slowly at as low a pressure as possible. No aperient is given, but atropine should be administered to counteract the spasm which is almost invariably present; gr. 1/100th three times before meals can be tried as an initial dose, which should be increased if no unpleasant degree of dryness of the mouth or ocular symptoms arise.

When the signs of inflammation have disappeared the volume of the paraffin enema can be slowly reduced, and finally paraffin given by mouth can be substituted for it, beginning with one ounce three times a day and gradually reducing the quantity till the right dose is found sufficient for producing one or two soft, unformed stools a day.

By treatment of this kind we have found it possible not only to cure the slight cases in which the question of surgery could hardly arise, but even to relieve severe cases, in which obstructive symptoms and an acutely tender inflammatory mass, accompanied by pyrexia and leucocytosis, were present. It is generally possible to avoid an operation in the acute stage, when it is most dangerous. If after the disappearance of urgent symptoms the treatment described under the head of prophylaxis is continued indefinitely, the patient can in many cases be kept permanently in good health without resorting to surgery. The following case is a good example of successful medical treatment of a severe case.

*Case of diverticulitis treated without operation.*—Mr. D., aged 60, was admitted into New Lodge Clinic on April 23, 1924, for constipation and severe abdominal pain. He had had dysentery in 1902 and suffered from intermittent attacks of diarrhoea during the following ten years. From 1912 his bowels had acted with perfect regularity until January 1924, when he

noticed his stools were much less formed than usual. He had been in the habit of having weekly abdominal massage; ten weeks before admission this began to cause pain in the left iliac fossa. During the last six weeks he had had slight intermittent pyrexia and began to feel generally unfit. From the middle of March he had increasing difficulty in getting his bowels opened, two or three days often passing without an action,



FIG. 8.

Diverticula of iliac and pelvic colon seen immediately after evacuation of the greater part of an opaque enema.

which was eventually obtained with an enema. On April 15 he had a sudden attack of very severe colicky pain in the lower abdomen, which was only partially relieved by hot applications. Though he felt a strong desire to open his bowels, he could pass neither fæces nor gas. His temperature was  $101^{\circ}$ . The attack passed off in three or four days, but left a continuous feeling of discomfort in the left iliac fossa. The bowels were now opened by daily enemata, but the patient noticed that whereas he formerly



took two and a half pints with ease, he now could only retain about a pint, and that the fluid felt as if it had to pass some obstruction.

On admission the iliac colon was found to be greatly thickened and extremely tender; there was a good deal of muscular rigidity over the left iliac fossa which made deep palpation difficult. The stools contained no obvious blood or mucus, though traces of occult blood were found by the guaiac test. The sigmoidoscope was passed ten inches without difficulty, and the mucous membrane looked perfectly healthy. A barium enema showed that from a point about midway along the pelvic colon to the junction of the descending with the iliac colon there was an obvious narrowing of the lumen, which widened again in the descending colon. The lumen was also very irregular and numerous diverticula could be recognised (Fig. 8). Some days later, when the acute symptoms had passed, a barium meal was given; there was now no delay in the passage through the colon, some barium being in the stools twenty-four hours after it was taken.

There was definite leucocytosis, the number of white cells being 15,400 per c.mm., of which 69 per cent. were polymorphonuclear cells. There was no anæmia, the red cells numbering just over 5,000,000 per c.mm. The temperature was irregular, rising to a maximum of  $101.2^{\circ}$  F. four days after admission. There had been no loss of weight. Rest in bed, careful dieting and paraffin enemas resulted in the rapid disappearance of active inflammation. At the end of a week the temperature was normal and the number of leucocytes had fallen to 10,400 per c.mm. The local signs slowly disappeared, and when the patient left the Clinic at the end of three weeks all rigidity and tenderness had gone. There was no longer any definite tumour in the left iliac fossa, though the iliac colon was still rather thicker than normal.

In the seventeen months which have elapsed since the patient left the Clinic he has led an active life and has remained absolutely free from symptoms. He continues to take the prescribed diet and two ounces of paraffin by mouth every night.

#### (b) SURGICAL TREATMENT

By R. P. ROWLANDS, M.S.

##### *Indications*

If the medical treatment already described does not lead to rapid improvement in obstructive or acute inflammatory cases, an operation should be performed without further delay. In more favourable cases, if in spite of careful after-treatment the symptoms recur, it is probably wise to operate as soon as they have again subsided, as in a quiet, afebrile period a radical

operation may be hopefully undertaken and completed at one sitting. In the very exceptional acute cases, in which the signs suggest a spreading peritonitis, immediate operation gives the only hope of saving life. In any case in which it proves difficult to exclude carcinoma, either primary or complicating diverticulitis, operation should also be advised.

#### CHOICE OF OPERATION

It is often difficult to decide upon the best treatment even when a correct diagnosis has been made either before or at the operation. Telling<sup>8</sup> has drawn attention to the fact that these patients are particularly susceptible to infection. Peritonitis and suppuration of the abdominal wall have followed in too many cases because of the lurking infection in, and especially around, the affected colon. It is very important to prepare the patient carefully and to take every precaution against infection. It is wise to defer radical operation for at least a month from the end of a febrile attack.

The following operations may be briefly discussed :

- (a) Excision of diverticulum.
- (b) Colostomy.
- (c) Short-circuiting.
- (d) Resection.
- (e) Closure of colico-vesical fistula.
- (f) Treatment of abscess and peritonitis.

##### (a) *Excision of Diverticulum*

Excision of one or more diverticula is indicated when the disease is limited to one or a few. It has been successfully performed both for acute and chronic diverticulitis, but symptoms are apt to recur after it, because other diverticula are often present but unperceived and these may become inflamed at any time.

##### (b) *Colostomy*

Colostomy affords much relief without great risk of life, but an artificial anus has so many obvious disadvantages as to make it undesirable for sensitive patients. As a temporary measure it may be tolerated, when primary resection or short-circuiting is not safe or is impracticable at the time. It is particularly objectionable for the permanent treatment of patients who are not suffering from malignant disease and may be expected to survive for many years. It is of the utmost importance to bear in mind that what appears to be an obvious

malignant growth of the pelvic colon may be only inflammatory. The mistake may be made even when the abdomen is opened. When operating for acute, following upon chronic, intestinal obstruction or when performing colostomy because a supposed growth is too adherent and extensive for removal, it may be useful to remove a small piece for microscopic examination. It must be remembered, however, that a good deal of inflamed tissue surrounds a growth, so that too much reliance must not be placed upon a negative report. Occasionally the rest, drainage and irrigation afforded by the colostomy may lead to partial or even complete disappearance of the stricture so that the artificial opening may be closed, but the trouble may recur. In other cases a temporary colostomy or valvular cæcostomy may be followed by secondary resection of the diseased part of the colon with restoration of the continuity of the bowel. In many cases this is the safest plan.

(c) *Short circuiting*

It is unfortunate that the disease is usually in a part of the colon which is difficult to short-circuit. Moreover, the anatomical relations are greatly altered by the disease. The meso-sigmoid is shortened and thickened and the inflammation may extend to the descending loop of the pelvic colon, so that it is almost impossible to make the anastomosis in healthy bowel below the disease: however, with the aid of a rubber tube, it may be possible to join the cæcum to the front of the rectum. In one case in which a tubular stricture extended from the rectum to the splenic flexure I anastomosed the ileum laterally to the front of the rectum, thus affording incomplete but considerable relief, the patient surviving in fair health for at least fifteen years. When the stenosis is high up, as, for instance, in the splenic flexure or descending colon, a short-circuit can be easily performed.

(d) *Resection*

Resection with end-to-end union appears to be the operation of choice, for it offers a good chance of cure at one operation without undue immediate risk. In some of these cases it is much easier and safer to make the end-to-end anastomosis with the aid of a large rubber tube inside the rectum and colon. It is necessary to prepare the patient carefully by dieting, aperients and enemata before undertaking the operation. It is wise to wait until any acute attack of inflammation has subsided. I have performed this operation in thirteen cases with one death from peritonitis; in this case the anastomosis, low in the pelvis, was unfortunately not made over a rubber tube.

The ultimate results have been very good. The two following cases are good examples of this condition.

*Resection of pelvic colon for diverticulitis, simulating carcinoma.*—Mrs. M. T., aged 59, had suffered from constipation all her life, but this had been worse lately. In 1922 she had an attack of abdominal pain which was thought to be due to kinking of the bowel; the pain was very severe, requiring an injection of morphia. The attack was accompanied first by diarrhœa, then by severe constipation: the temperature was above normal for a fortnight. Two weeks later another attack developed, accompanied by fever and shivering, a quick pulse and severe abdominal pain, which was worse in the lower abdomen. There was another attack in May 1924. I saw the patient in March 1925, when a tumour could be felt high up in the posterior wall of the rectum. It was thought to be a carcinoma of the dependent loop of the pelvic colon, lying in Douglas' pouch. It was not adherent either to the uterus or to the sacrum. An operation was advised.

*Operation, March 25, 1925.*—Left low paramedian incision. Extensive diverticulitis of the pelvic colon with many small stones, a little larger than peas, in diverticula at the side of the colon. The bowel and its mesentery were shortened from pericolitis and infiltration. The whole of the pelvic colon, the lower part of which was stenosed, was excised and end-to-end union made over a tube. There was no sign of growth. There were numberless diverticula, even the descending colon above the resection showed a few small pouches.

The patient made a good recovery and was in excellent health six months later.

*Resection of pelvic colon for diverticulitis simulating carcinoma.*—Mr. E. W. had suffered during the last year from three attacks of intestinal obstruction, each lasting four or five days. Each attack was associated with severe griping pains and abdominal distension: enemata ultimately brought relief. Radiographic examinations of the colon in the intervals failed either to show the cause of the obstruction or to give evidence of diverticulitis. When I saw him in the last attack the transverse colon was seen to be dilated and contracting violently at intervals. The left iliac colon was in a similar condition. There was no history of melæna. Carcinoma of the pelvic colon was strongly suspected and an operation was performed four days after the obstruction had been overcome by enemata and liquid paraffin. A mass, three inches long, was found in the lower part of the pelvic colon, adherent to the left ureter. It was removed with considerable difficulty owing to the thickening and shortening of its mesentery and the adhesion to the side and back of the pelvis. An end-to-end union was made over a large rubber tube which drained the descending colon through the rectum and anus. The patient made a good and rapid recovery.

After the mass had been removed it was still thought to be a carcinoma, but on slitting up the bowel no growth was found, only a light, fibrous, tubular stricture, two and a half inches long, due to chronic inflammation around numerous diverticula. It was difficult to introduce the blade of a pair of scissors into the stricture.

In some cases immediate resection with drainage of the bowel above and below, after Mikulicz' and Paul's method, has been successfully done, Paul's tubes being tied into the cut ends of the remaining colon. Another operation is generally necessary to re-establish the natural channel, although this may take place naturally by the sinking in of the colostomy openings.

(c) *Closure of colico-vesical fistula*

When a fistula has developed between the colon and the bladder, as a result of diverticulitis of the colon, and the diagnosis of non-malignant disease has been established by means of the cystoscope and x-rays, an attempt may be made to cure this very troublesome and dangerous condition by laparotomy. The sigmoidoscope, which must be used with great care, may reveal the site of adhesions, stricture and perhaps of the fistula. If the lower six inches of the bowel can be proved to be healthy, an operation is hopeful. If the obstruction and fistula are lower, the operation may be very difficult and dangerous. Detachment of the colon from the bladder, with closure of the openings into these viscera, may be performed with the aid of the Trendelenburg position and a good light. The opening into the bladder is fairly easily closed and inverted and, if the disease in the bowel is fortunately localised without stenosis, a similar plan may be adopted for it. If, however, the disease is extensive and causes a definite stricture, and especially if adhesions are dense and extensive, preliminary colostomy with spur formation is indicated. This relieves the distressing pain and cystitis and greatly improves the general condition of the patient. It also makes the subsequent resection of the diseased part of the colon much safer. It is sometimes wise to make the colostomy only a little above the stricture so that it can be removed and the channel of the bowel re-established at the second operation. In some bad cases permanent colostomy is the only safe treatment. The following is an interesting example.

*Resection and colostomy for vesico-colic fistula secondary to diverticulitis.*—Mrs. G., aged 55, had a laparotomy six years before by a gynaecologist for pelvic tumour: a swelling was found which was thought to be carcinoma of the pelvic colon.

There was no obstruction at the time, therefore the growth was replaced and the abdomen closed with the idea that a surgeon should remove the growth later, but the wound suppurated and the patient declined to have another operation. Apart from constipation, from which she had suffered for many years, she was comparatively well for the next six years, but in February 1920 she had signs of inflammation in the lower part of the abdomen and a large swelling formed in the left iliac fossa. There was high fever of the remittent type. An operation was advised but declined. Three weeks later the abscess apparently ruptured into the bladder, and after this she often passed gas and pus with the urine and the temperature continued high and remittent. She lost flesh, became anæmic and at last consented to see a surgeon. I saw her on April 12, 1920, and persuaded her to have an operation. She went into a nursing home, but was so much better for a week that the operation was deferred. However, her temperature went up again and the bladder symptoms continued so that the operation was performed. Diverticulitis of the colon with fistula in the bladder was diagnosed.

*Operation, April 26, 1920.*—Vertical incision over the lower half of the left rectus. Many dense adhesions in the pelvis between the pelvic colon and bladder. On separating these several abscesses were opened and two cysts, containing sanious fluid, near the left ovary and broad ligament. The left Fallopian tube contained pus and was removed. The fistula into the bladder was found well to the left of the middle line; the opening was closed with catgut and this part was then pushed down in front of the uterus, which was sewn up over it.

The opening on the front of the pelvic colon communicating with the bladder would admit a lead pencil, and its mucous membrane prolapsed and was clearly part of a diverticulum. There was another similar opening lower down and to the right, which was found on trying to separate the lower part of the pelvic colon. The latter was very much shortened, thick, hard and intimately adherent back and front. Its wall was so rigid that it appeared practically impossible to close the openings mentioned, therefore the diseased part was separated and excised. The disease extended too low to make it possible to join the descending colon to the rectum, therefore the cut end of the rectum was closed and inverted and an end colostomy made at the upper end of the wound.

The specimen removed was about eight inches long; its lumen was considerably narrowed. The openings of many diverticula were found on its mucous surface, many of them hidden by folds of mucous membrane, some extending into fairly large pouches in the fat at the side of the colon.

The patient made a slow but good recovery: the cystitis and shivering ceased at once but the wound suppurated for some weeks. The restoration of the natural passage was suggested

eighteen months later, but the patient preferred to carry on as she was, the colostomy causing her but little inconvenience.

(f) *Treatment of Abscess and Peritonitis*

Acute diverticulitis of the colon is best treated conservatively, for it rarely causes diffuse or spreading peritonitis. The patient is placed in the Fowler position and is given nothing by the mouth until the fever has subsided. Saline infusions are given if necessary. If an abscess forms, it is opened and drained through an incision above and to the left of the pubes. If the pulse, temperature, pain and abdominal rigidity increase, indicating spreading peritonitis, in spite of treatment, the abdomen is opened and any perforation found is closed, the perforated diverticulum being removed if possible.

SURGICAL MORTALITY

Cases of diverticulitis (16) at the Mayo Clinic from January 1, 1907, to January 1, 1924 (Judd and Pollock).

Type of operation.	Number of patients.
Mikulicz . . . . .	35
Tube resection . . . . .	12
Resection . . . . .	21
Colostomy . . . . .	15
Excision of the diverticulum . . . . .	15
Drainage of abscess . . . . .	7
Anastomosis . . . . .	7
Exploration . . . . .	6
Total . . . . .	118

The mortality of these operations was about 10 per cent.

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## ON THE SIGNIFICANCE OF COLIFORM BACILLI IN THE DUODENUM

By F. A. KNOTT, M.D., Pathologist to New Lodge Clinic.

IN making bacteriological examinations of samples of duodenal contents and gall-bladder bile withdrawn through an Einhorn tube, the frequency with which strong growths of coliform bacilli can be obtained appears to be in sharp contrast with the percentage occurrence in saliva and sputum of this group of organisms. It has been attempted, therefore, in the following analysis to ascertain what clinical deductions, if any, can be drawn from a determination of the exact biological group and pathogenicity of these bacteria.

Organisms in specimens obtained by the Einhorn tube are all to some extent suspect of being derived from salivary contamination, and to minimise this uncertainty the technique of collecting specimens outlined in a previous communication<sup>1</sup> has again been rigidly adhered to in the present series. On that occasion reference was made to the importance of recognising crystals, pigment granules and leucocytes in the duodenal contents, and the point is again illustrated by these further cases (see Table I), but it will not be discussed in this note.

### COLIFORM BACILLI IN SALIVA

It will be best first to consider the frequency with which normal saliva may contain coliform organisms. Glynn and Digby<sup>2</sup> found that among 125 normal salivas, 6.4 per cent. contained coliform organisms. Reference to my own records of 175 samples of saliva and non-purulent sputum obtained from "normals" or patients suffering from nothing beyond mild catarrhal states of the upper respiratory tracts shows coliform bacilli to be present in 7.4 per cent. Combining the two series (300 cases), the case incidence is approximately 7 per cent. The strains most frequently isolated belonged to the *B. Friedländer* group; *B. coli communis* and organisms of the typhoid-dysentery groups were never found.

### CASE INCIDENCE IN DUODENAL CONTENTS

In contrast with the above figures, it may be noted that in 172 routine examinations of gall-bladder bile obtained by



duodenal tube from patients under the care of Dr. A. F. Hurst in Guy's Hospital and New Lodge Clinic, coliform bacilli have been found in quantity in 65, *i.e.* approximately 87 per cent. Assuming that in even 10 per cent. of these people the organisms might arise from salivary contamination, in 27 per cent. they must in all probability represent an essential part of the duodenal flora. It has not been possible to determine the exact biological grouping of the bacteria in all the above 65 cases, as the cultures from the earlier specimens were, at the time the present tests began, no longer available. Thirty-six have, however, been examined, and the results are summarised below.

#### CLASSIFICATIONS ADOPTED

The methods used in classification of the bacilli largely involve routine laboratory procedure and need be given in outline only.

Note was made of the following points concerning each strain :

(1) General characters and nature of growth on artificial media including gelatin.

(2) The fermentative action upon glucose, lactose, saccharose, maltose, mannite and dulcitol; the indol and litmus-milk reactions.

(3) In cases recorded as "unclassified," guinea-pigs were inoculated intraperitoneally with 1 c.c. of a 24-hour broth culture of the bacillus. None of these strains was found to be pathogenic to these animals.

Among those coliform bacilli which fermented lactose, MacConkey's<sup>3</sup> classification was adopted and the organisms placed in one of the following groups :

(A) *B. acidi lactici* group.

(B) *B. coli communis* group.

(C) *B. pneumoniae Friedländer* group.

(D) *B. lactis aerogenes* group.

(E) *B. proteus*.

For those bacilli which did not ferment lactose there is no universally accepted classification, but the following types may serve the purpose of this analysis :

(F) Typhoid-Paratyphoid groups.

(G) Dysentery Group.

(H) Morgan's group.

(K) *B. faecalis alkaligenes*.

(L) Unclassified gelatin-liquefying non-pathogenic strains.

(Subsequently these groups may be conveniently referred to simply by the prefixed capital letters.)

## PATHOGENICITY OF COLIFORM BACILLI

The high pathogenicity of some of the above groups is well recognised, whereas others are known rarely to cause lesions. It is impossible to fix any absolute line of division, and upon this point only broad generalisations can be made. Nevertheless the pathogenicity of Groups B, C, D, F and G would appear to be sufficiently well recognised. Glynn and Digby<sup>2</sup> failed to find any evidence of pathogenicity for organisms of Group A. Organisms in Groups E, H, K and L are generally considered to be of low pathogenic power,<sup>4</sup> with the exception, perhaps, of Morgan's No. 1 bacillus and some strains of *B. proteus*.

Therefore, in this note, an arbitrary division of the organisms is made into one set having established pathogenicity (Groups B, C, D, F and G), and another in which the numbers are usually less harmful and frequently encountered as simple saprophytes (Groups A, E, H, K and L).

## ANALYSIS OF CASES SHOWING DUODENAL COLIFORM BACILLI

In summarising the findings in the 36 cases examined it is found that they fall naturally into two classes: (1) those showing definite or probable lesions of the biliary tracts, and (2) those with no signs of this condition, but having in most cases very low gastric acidity. Thus in Table I are given details of 16 cases of biliary tract disease, and in Table II the corresponding findings in 20 cases of other pathological conditions.

In most of these cases bacteriological examination of the sputum or saliva was made. None of the specimens contained coliform bacilli.

As far as the exact strains of coliform bacilli are concerned, it will be noted that among the 16 cases of biliary tract lesions (Table I), organisms of Groups B, C, D, F and G (the "pathogenic" strains) occur in 15 cases and organisms of Groups A, E, H, K and L (the "saprophytic" strains) in one. Moreover, in 14 cases the test-meal result is normal; in one there is hyperchlorhydria and in one achlorhydria.

Among the 20 cases of "other diseases" (Table II), the organisms of Groups B, C, D, F and G occur in 6, those of A, E, H, K and L in 14. In these cases the gastric acidity is low in 15, normal in 3, and hyperchlorhydria occurs twice.

## CONCLUSIONS

On comparing these tables it is evident that the definitely pathogenic types of coliform bacilli occur more frequently in the cases showing biliary tract lesions, approximately 90 per

TABLE I.

No.	Patient.	Duodenal contents after Mag. sulph.		Gall-bladder at operation.	Clinical diagnosis and Remarks.	Test-meal result.
		Crystals.	Leucocytes.			
1	Mrs. V.	Nil	—	No operation	Typical signs of cholecystitis	Normal curve
2	Mr. S.	Nil	—	No operation	Typical signs of cholecystitis	Normal curve
3	Mrs. C.	Pigment and cholesterol	—	Three pigment stones and chronic cholecystitis	<i>B. typhosus</i> grown from gall-bladder bile and stone 23 years after typhoid fever	Normal curve
4	Mr. S.	Nil	+	No operation	Signs of cholecystitis	Hyperchlorhydria
5	Mrs. H.	Nil	—	No operation	Signs of cholecystitis	Normal curve
6	Lady T.	Pigment granules	—	No operation	Signs of cholecystitis	Achlorhydria
7	Mrs. W.	Nil	—	Gall-stones and chronic cholecystitis	Stones and gall-bladder infected with <i>B.c.c.</i>	Normal curve
8	Mrs. B.	Nil	+	Gall-stones	Gall-stones at operation	Normal curve
9	Mrs. L.	Nil	also red cells	No operation	Typical cholecystitis	Normal curve
10	Mrs. B.-W.	Nil	+	No operation	Definite cholecystitis	Normal curve
11	Mrs. R.	Nil	—	No operation	Definite cholecystitis	Normal curve
12	Mrs. E.	Nil	—	No operation	Signs of cholecystitis	Normal curve
13	Rev. B.	Pigment granules	—	Gall-stones	Gall-stones at operation	Normal curve
14	Mrs. B.	Nil	—	Gall-stones	Gall-stones at operation	Normal curve
15	Miss R.	Pigment granules	+	No operation	Clinically gall-stones but no operation	Normal curve
16	Mr. R.	Nil	—	No operation	Blood agglutinated <i>B. typhosus</i> strongly. Typhoid peritonitis with no sign of cholecystitis 10 years after typhoid fever	Normal curve

TABLE II.

No.	Patient.	Duodenal contents after Mag. sulph.			Clinical diagnosis and Remarks.	Test-meal result.
		Crystals.	Leucocytes.	Coliform strain.		
1	Mrs. B.	Nil	Nil	<i>B. fecalis alkaliogenes</i>	Achalasia of the cardia	Achlorhydria
2	Miss C.	Nil	Nil	Unclassified	Post-influenzal debility	Hypochlorhydria
3	Mrs. E.	Nil	Nil	<i>B. lactis aerogenes</i>	Septic anemia	Very low acid
4	Miss F.	Nil	Nil	Unclassified	Anemia of septic type	Achlorhydria
5	Miss G.	Nil	Nil	Unclassified	Secondary anemia	Very low acid
6	Mrs. H.	Nil	Nil	<i>B. Friedländer</i>	Duodenal ulcer	Hypochlorhydria
7	Mr. H.	Nil	Nil	<i>B. coli communis</i>	Cirrhosis and alcoholic gastritis	Achlorhydria
8	Mrs. K.	Nil	Nil	<i>B. acidi lactici</i>	Fibrositis and neurasthenia	Achlorhydria
9	Mr. R.	Nil	Nil	<i>B. proteus alkaliogenes</i>	Dyschezia	Normal
10	Mr. R.	Nil	Nil	<i>B. fecalis alkaliogenes</i>	Subacute gastritis	Achlorhydria
11	Dr. S.	Nil	Nil	<i>B. acidi lactici</i>	Fibrositis	Low acid
12	Col. S.	Nil	Nil	Unclassified	Rheumatoid arthritis	Low acid
13	Mrs. B.	Nil	Nil	<i>B. lactis aerogenes</i>	Addison's anemia and subacute combined degeneration of cord	Achlorhydria
14	Miss G.	Nil	Nil	<i>B. coli communis</i>	Gastritis: abuse of purgatives	Hypochlorhydria
15	Mr. H.	Nil	Nil	Unclassified	Anaemia and gastric carcinoma	Achlorhydria
16	Mrs. H.	Nil	Nil	<i>B. acidi lactici</i>	Fibrositis and neurasthenia	Normal
17	Mr. I.	Nil	Nil	Unclassified	Addison's anemia	Very low acid
18	Mr. M.	Nil	Nil	<i>B. coli communis</i>	Tuberculous ileo-colitis	Normal
19	Mrs. S.	Nil	Nil	Unclassified	Visceropitosis; paroxysmal tachycardia	Hypochlorhydria
20	Mr. S.	Nil	Nil	<i>B. fecalis alkaliogenes</i>	Alcoholic gastritis	Very low acid

cent. in Table I as compared with 80 per cent. in Table II. Nevertheless, as examples of both groups of bacilli occur in both tables, it is clear that one can rarely decide simply from the biological grouping of the organism isolated from bile-containing duodenal contents, whether it is producing or likely to produce inflammatory lesions. An essential factor in the correct interpretation of the bacteriological report must be consideration at the same time of the result of gastric analysis and the bacteriology of the saliva.

If the coliform strain isolated from the duodenum be found also in the saliva its significance may obviously be small. It is, however, clear from the figures given that these duodenal coliform bacilli are but rarely derived from salivary contamination.

From Table II it seems that, if the gastric juice is deficient in HCl, coliform bacilli of any strain may become permanent inhabitants of the duodenum and, even though they be of potentially pathogenic type, may do little harm to the patient. It may here be recalled that in our earlier series <sup>1</sup> a high percentage (85 per cent.) of bile-containing duodenal contents obtained from persons without biliary tract lesions, and having even moderate quantities of free HCl in the gastric juice, showed no infection.

Table II appears clearly to indicate that if a coliform bacillus can be grown in quantity from the gall-bladder bile of a person giving a normal fractional test-meal result then serious consideration must be given to the possible presence of a biliary tract lesion. Also one might suggest that, in such cases, although the bacilli isolated belong to strains the pathogenicity of which is usually considered to be low, this fact does not necessarily negative the possibility of the organism isolated being the cause of the patient's lesions.

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## A NOTE ON THE TREATMENT OF ACTIVE RICKETS BY THE MERCURY-VAPOUR LAMP

By J. F. CARTER-BRAINE, M.B., Chief Clinical Assistant, Actino-therapeutic Department, and A. A. OSMAN, D.S.C., Chief Clinical Assistant for Diseases of Children, Guy's Hospital.

DURING the past eighteen months we have had about thirty cases of active rickets undergoing treatment with the mercury-vapour lamp in the Light Department of this hospital. The greatest difficulty we had to face was irregularity in attendance. As a result of this we feel only justified in recording here the results obtained in eleven cases. Although most of the mothers were quite enthusiastic about the treatment, it must be remembered that many of them had to earn a living in addition to attending to the family, and so were unable to attend more than once or twice in the week, whereas daily attendance is required to secure the best and quickest results. The children were taken from the Children's Out-Patient Department and attended daily, whenever possible, for the treatment. It may be objected that the children were living at home, under more or less unknown conditions of diet and hygiene. This could not be avoided, as it was obviously not possible to admit them all into hospital. A few cases from the Children's Ward were treated, but they cannot be used for comparison with those living under home conditions.

### GENERAL PROCEDURE

It is necessary to point out, first, that all the cases were acute, or, more accurately, active. The term "acute" is misleading when applied to a process which is, or may be, continuously active over a period of weeks or months. The activity was estimated clinically by such signs as muscular hypotonia, sweating, pallor, and the usual deformities. Most of the cases were also slightly pyrexial; this is quite usual in the active stages of the disease. Further confirmation was obtained by x-ray examination of the epiphyses of the wrists, knees and ankles. Though we are well aware that in rickets the muscular system, and probably the whole of the body, suffers just as much as the bony system, we have expressed our results chiefly in terms of the x-ray appearances of the latter, because of the facility with which x-ray photographs could be taken at intervals and used

for comparison in the estimation of the progress made. Radiogram plates were taken every fourteen days.

#### DETAILS OF MERCURY-VAPOUR LAMP TREATMENT

The actual exposure to the light was performed in the following manner. At the commencement of the investigation a gradually increasing area of the child's body, beginning with the legs below the knees, was exposed to the rays for five minutes, at a distance of one metre from the lamp. After three applications the surface exposed was increased so as to include the thighs, and, after another three exposures, the area was again extended to include the abdomen. In this manner the whole of the body was very gradually exposed to the rays. This method was abandoned later, as it was found to be unnecessary, and thereafter we exposed the whole of the body except the face from the beginning of treatment, and simply increased the time of exposure. The children were placed naked on a rug inside a wooden pen with the eyes protected by tinted goggles. For the first week the child was exposed to the radiations for five minutes daily, and each successive week the dosage was increased by five minutes up to a maximum of fifty minutes daily. This exposure was attained in about ten weeks. The distance of the child from the lamp was one metre throughout. We found it advisable to have the lamp placed at the side of the child rather than directly overhead as in one instance the quartz container cracked and allowed the hot mercury to escape, though fortunately the child was not injured.

#### GENERAL RESULTS

Although we have estimated the results of treatment by the x-ray appearances of the bones, we were much impressed by the rapidity with which the general health of the children improved. Especially was this the case in regard to the muscle tone. In all of the cases before treatment muscular hypotonia was most marked, and in several instances the children could not sit up and would lie almost motionless, quite unable to move the limbs or take any interest in their surroundings. Generally at the end of ten to fourteen days' treatment, the physical condition and the muscular tone had so improved that they would be able to sit up and play. This improvement in the general condition appears to precede recognisable x-ray changes in the bones in every case.

Pigmentation occurred in only two of our cases (No. 2 and No. 9) after  $8\frac{1}{2}$  and  $9\frac{1}{2}$  hours' exposure respectively, that is at a time when the healing process in the bones was already well advanced.

RESULTS OF THE MERCURY-VAPOUR LAMP TREATMENT OF RICKETS

No.	Name.	Age in months.	X-ray condition before treatment.	Treatment commenced.	Number of weeks before first sign of improvement.	Number of hours exposure before first sign of improvement.	Total number of applications.	Total dosage in hours.	Pigmentation after hours' exposure.	Attendance.
1	Charles C.	19	Very advanced; active.	1.5.24	5	6½	39	11½	—	Good.
2	Elsie S.	18	Advanced; slightly active.	8.5.24	4	2	54	13½	8½	Good.
3	Julia B.	36	Advanced; very active.	6.7.23	2	1½	38	15½	—	Good.
4	Donis S.	16	Advanced; very active.	17.7.23	3½	2½	15	3½	—	Good.
5	Leonard M.	21	Advanced; very active.	24.7.23	No change.	No change.	18	3½	—	—
6	Leonard S.	18	Active.	28.12.23	13	5½	23	6½	—	Very irregular.
7	Arthur S.	24	Active.	25.1.24	12	20	109	63½	—	Fair but irregular.
8	Albert B.	21	Advanced; active.	14.2.24	No improvement.	No improvement.	21	7½	—	Very irregular with long intervals.
9	Joan J.	15	Advanced; active.	5.4.24	5	6	36	15½	9½	Good.
10	William J.	24	Well marked; active.	19.8.24	5	2½	26	10½	—	Irregular.
11	Maurice E.	21	Slightly active.	20.8.24	5	2	25	2	—	Fair.



## CHANGES IN THE BONES

The earliest x-ray appearances showing definite improvement was in Case 3 after  $1\frac{1}{2}$  hours' total exposure, and similar changes were noted in Cases 2 and 4 after 2 and  $2\frac{1}{2}$  hours respectively. On the other hand, Cases 5 and 8 showed no improvement after  $3\frac{1}{4}$  and 4 hours' treatment respectively, but Case 8 was very irregular in attendance. Case 7 only showed slight improvement after 20 hours. The average time at which healing was first observed was a little under 7 hours of actual exposure to the rays. Reference to the Table renders further detailed discussion of the cases unnecessary. The columns headed "Total Dosage in Hours" and "Total Number of Applications" have been added to give some idea of the regularity or otherwise of attendances.

## SUMMARY

It must be remembered that rickets is a self-limited disease, and that spontaneous recovery with or without a certain amount of deformity is the rule, whatever line of treatment is adopted. The comparative value of any form of treatment for this disease is measured rather in the rapidity with which the normal process of healing can be accelerated than in anything else. No comparison can be made between our results and those of other workers either with this or with other methods of treatment, as our cases were not under standard conditions of diet and hygiene.

Dr. Chick <sup>1</sup> and her colleagues in Vienna, for example, found that exposure to direct sunshine, or to the ultra-violet rays from the mercury vapour quartz lamp, or the administration of cod-liver oil, would all bring about the onset of healing, as judged radiographically, in from two to four weeks, all the cases being under standard conditions. In the absence of control cases we do not feel disposed to offer any comment upon the results obtained so far, beyond stating that the method appears to be valuable and to warrant further trial.

In conclusion we should like to express our thanks to Mr. Magnus Redding for his valuable assistance with the radiographic work, to Dr. C. E. Iredell for permission to conduct the investigation in his department, and to Dr. H. C. Cameron, from whose Out-patient Department the cases were obtained.

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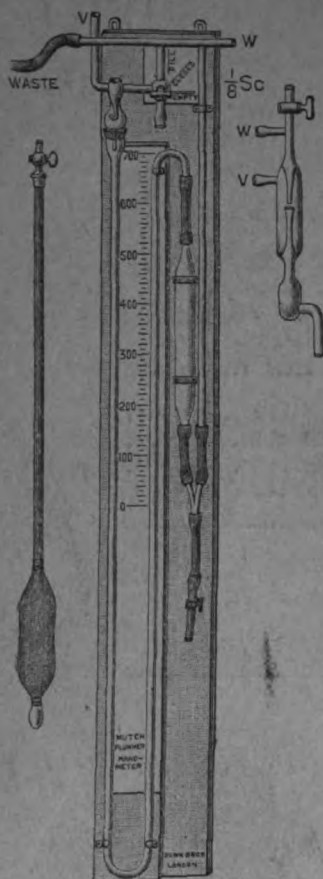
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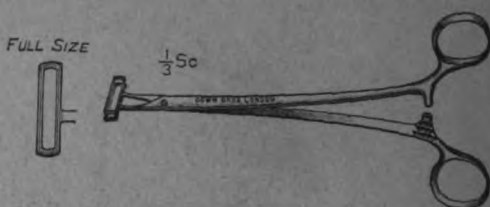
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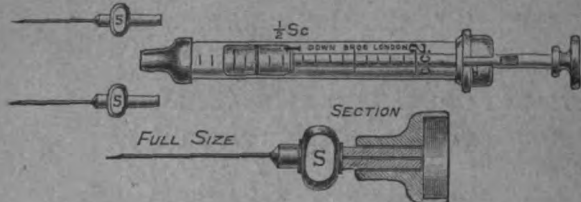
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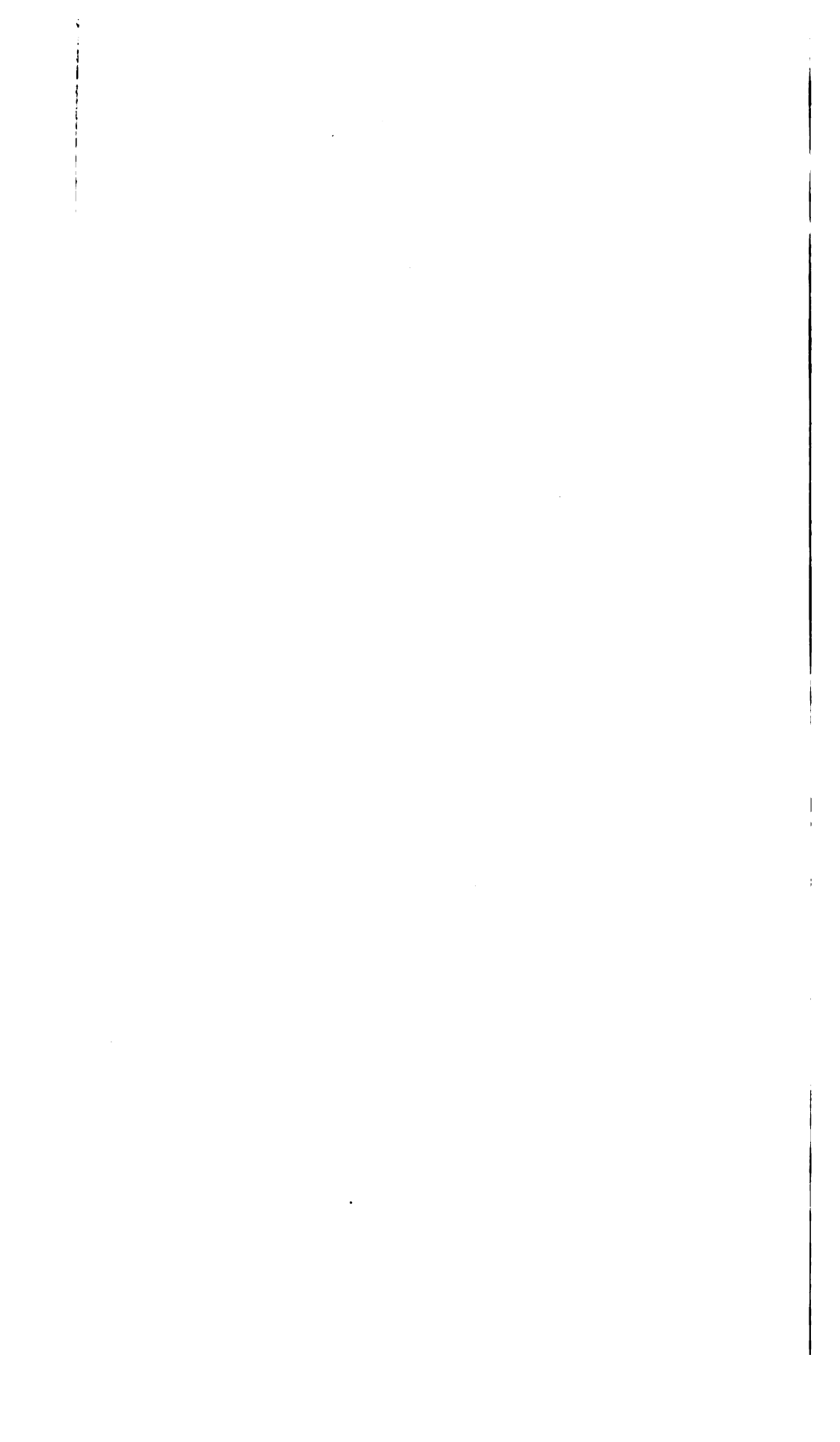
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